The American Journal of Surgery

PUBLISHED MONTHLY BY THE AMERICAN JOURNAL OF SURGERY, INC.

49 WEST 45TH STREET, NEW YORK, N. Y.

Editor: THURSTON SCOTT WELTON, M.D., F.A.C.S., NEW YORK

EDITORIAL BOARD

DONALD C. BALFOUR, *Rochester, Minn.*; CARL BECK, *Chicago*; GEO. R. BRIGHTON, *N.Y.*; MEREDITH F. CAMPBELL, *N.Y.*; JAMES T. CASE, *Chicago*; ISIDORE COHN, *N.O.*; BRADLEY L. COLEY, *N.Y.*; FREDERICK A. COLLER, *Ann Arbor*; GEORGE W. CRILE, *Clev.*; ELLIOTT C. CUTLER, *Boston*; ROBERT V. DAY, *Los Angeles*; CHARLES A. ELSBERG, *N.Y.*; HERBERT C. FETT, *Brooklyn*; C. R. G. FORRESTER, *Chicago*; H. DAWSON FURNISS, *N.Y.*; JOHN H. GIBBON, *Phila.*; EMIL GOETSCH, *Brooklyn*; CHARLES A. GORDON, *Brooklyn*; DONALD GUTHRIE, *Sayre, Pa.*; A. E. HERTZLER, *Kansas City*; LOUIS J. HIRSCHMAN, *Detroit*; J. M. HITZROT, *N.Y.*; FREDERICK C. HOLDEN, *N.Y.*; EMILE F. HOLMAN, *San Francisco*; J. A. HYAMS, *N.Y.*; R. H. JACKSON, *Madison*; JOHN E. JENNINGS, *Brooklyn*; W. L. KELLER, *Washington*; HOWARD A. KELLY, *Baltimore*; T. J. KIRWIN, *N.Y.*; ARTHUR KRIDA, *N.Y.*; A. V. S. LAMBERT, *N.Y.*; MAURICE LENZ, *N.Y.*; H. H. M. LYLE, *N.Y.*; JEROME M. LYNCH, *N.Y.*; URBAN MAES, *N.O.*; HARRISON S. MARTLAND, *Newark, N.J.*; RUDOLPH MATAS, *N.O.*; ROY D. MCCLURE, *Detroit*; H. C. NAFFZIGER, *San Francisco*; CLARENCE R. O'CROWLEY, *Newark, N.J.*; LOUIS E. PHANEUF, *Boston*; EUGENE H. POOL, *N.Y.*; DOUGLAS QUICK, *N.Y.*; N. P. RATHBUN, *Brooklyn*; HUBERT A. ROYSTER, *Raleigh*; HENRY S. RUTH, *Phila.*; A. C. SCOTT, *Temple, Tex.*; M. G. SEELIG, *St. Louis*; J. BENTLEY SQUIER, *N.Y.*; H. J. STANDER, *N.Y.*; GEORGE W. SWIFT, *Seattle*; H. L. UPDEGRAFF, *Los Angeles*; GRANT E. WARD, *Balt.*; J. H. WOOLSEY, *San Francisco*.

Foreign Collaborators—GREAT BRITAIN—J. H. WATSON, *Burnley*. FRANCE—G. JEANNENEY, *Bordeaux*. ITALY—R. BASTIANELLI, *Rome*.

NEW SERIES, VOLUME XLII

OCTOBER TO DECEMBER

1938

THE AMERICAN JOURNAL OF SURGERY, INC., PUBLISHERS

NEW YORK MCMXXXVIII

COPYRIGHT, 1938
BY THE AMERICAN JOURNAL OF SURGERY, INC.

All Rights Reserved

Printed in the United States of America

CONTENTS OF NEW SERIES, VOLUME XLII

ORIGINAL ARTICLES

The Use of the Oribicularis Palpebrarum Muscle in Surgery of the Eyelids	*John M. Wheller*	7
Surgery of Secondary Glaucoma	*Allen Greenwood*	10
The Technique of Extraction of Intraocular Foreign Bodies	*Walter B. Lancaster*	14
Technique of Scleral Fixation of Extraocular Muscles	*P. Chalmers Jameson*	25
Present Status of Tendon Transplantation of the Ocular Muscles	*Luther C. Peter*	30
Certain Postoperative Complications of Cataract Operations. With Especial Reference to a Study of 1,004 Operations	*Conrad Berens* *Donald W. Bogart*	39
Management of the Complications of Intraocular Surgery	*Everett L. Goar*	62
The Conjunctival Flap in Ophthalmic Surgery	*John Green*	69
Ectropion and Entropion of the Eyelids	*Webb W. Weeks*	78
The Surgical Technique in Tenotomy of the Inferior Oblique Muscle	*James Watson White*	83
A Review of some Modern Methods for Ophthalmic Plastic Surgery	*Edmund B. Spaeth*	89
Aural Surgery. Theoretic and Technical Advances in Recent Decades	*Philip D. Kerrison*	103
Acute Encephalitis or Toxic Encephalopathy Simulating Brain Abscess	*Paul J. Zentay*	112
Sinus Thrombosis	*I. Friesner* *J. G. Druss* *J. L. Goldman* *H. Rosenwasser*	116
The Diagnosis and Treatment of Otogenic Meningitis	*Samuel J. Kopetzky*	131
The Radical Mastoid Operation	*John Mackenzie Brown*	142
The Education of the Deaf and Hard-of-Hearing Child	*Max A. Goldstein*	151
On the Diagnosis and Treatment of Otosclerosis	*Louis K. Guggenheim*	156
Malignant Diseases of the Paranasal Sinuses	*Gordon B. New*	170
Dietary Treatment of Chronic Sinusitis	*Burt R. Shurly*	174
On the Application of Zinc Sulfate Solution in the Prevention of Poliomyelitis	*Edwin W. Schultz*	178
Applied Physiology of the Nose and the Accessory Nasal Sinuses	*Arthur Proetz*	190
Pathways of Referred Pain from the Nose	*Ralph A. Fenton*	194
External Operations on the Frontal Sinus	*Harold I. Lillie*	199
Deep Abscess of the Neck. Surgical Treatment	*John F. Barnhill*	207
Oral Lesions in Otolaryngology	*Dean M. Lierle* *Ruben Nomland*	220
The Use of Physical Therapy in Diseases of the Ear, Nose and Throat	*J. L. Myers*	232

Types of Laryngeal Obstruction and Their Treatment	*Lyman Richards*	239
The Treatment of Stricture of the Esophagus	*Clyde A. Heatly*	260
The Surgical Indications in Perforation of the Esophagus by Foreign Bodies	*Jerome R. Head*	266
Benign Tumors and Tumor-Like Conditions in the Tracheobronchial Tree	*Chevalier Jackson.* *Chevalier L. Jackson*	275
Sarcoma of the Uterus.	*J. Kotz* *Morton S. Kaufman*	289
A Simple, More Complete Obstetrical Chart	*Harry S. Fist*	294
Complications of the Weak Foot (Subtalar Dystrochoides)	*James Graham*	299
Muscular Rheumatism and Spina Bifida Occulta	*R. J. Dittrich*	318
An Analysis of the Standard Methods of Treating Fracture of the Femur. Based on an Analysis of 100 Consecutive Cases	*Denver M. Vickers*	332
Infection and Gangrene of the Extremities in the Diabetic. Diagnosis and Treatment.	*Manuel Grodinsky*	339
Hypervitamin Therapy in Surgical Practice	*Martin G. Vorhaus*	350
Pectenosis and Pectenotomy in Pruritus Ani. Preliminary Report	*Manuel G. Spiesman*	356
Misgivings on Mineral Oil as a Laxative	*James W. Morgan*	360
Mandelic Acid: Its Therapeutic Value and Limitations	*Grayson Carroll* *Bransford Lewis* *Louis Kappel*	365
Observation on Sulfanilamide in Urology	*Frederick A. Reuter*	368
Nephroptosis	*Jarratt P. Robertson* *Paul L. Singer*	379
Surgical Indications in Thyroid Disorders	*B. A. Goodman*	387
Ingrown Toe-Nail. Operation by Electrosurgery	*Sidney Vernon*	396
Chronic Appendicitis. Appendectomy in Absence of History of Acute Appendicitis or of Appendiceal Colic	*Manfred Kraemer*	398
Foreign Body in the Myocardium	*Benjamin H. Neiman* *J. E. Fitzgerald*	401
Subcutaneous Traumatic Rupture of the Pancreas Complicated by High Intestinal Fistula, Report of a Case	*David H. Smith*	406
Carbuncle of the Kidney: Report of Two Cases	*Harold E. Stedman*	410
Embolism Following Thyroidectomy for Hyperthyroidism	*George N. J. Sommer, Jr.* *Louis Levin*	415
Tuberculosis of the Lip	*Christopher Stabler*	419
Multiple Hernia. Case of Incarcerated Umbilical Hernia Associated with Spigelian, Epigastric, Ventral, and Mesocolic Hernias	*Harold A. Goldberger* *Richard R. Panebianco*	423
The Diagnosis of Acute Hernial Appendicitis. With Reports of Two Cases Occurring at Both Extremes of Life	*Thomas O. Burger* *Harold C. Torbert*	429
Report of a Case of Regional Ileitis	*J. James Cancelmo*	433

Bowen Speculum Cup	*Arthur Bowen*	435
A New Triplicate Colon Clamp after De Martel	*William L. Wolfson*	437
New Splint for Pelvic Fractures	*Joseph K. Narat*	443
Some Early Autopsies in Georgia	*Joseph Krafka, Jr.*	445
Technique of Thyroidectomy	*Arthur E. Hertzler*	449
Fundamental Philosophy of Workmen's Compensation	*Henry D. Sayer*	483
Medical Reports Bore Me, Too	*G. Lester Marston*	490
Medical Experts as Witnesses	*Eugene O'Dunne*	501
The Examination of a Patient for Medicolegal Purposes	*Frank L. Flack*	505
Conservative Treatment of Cerebrocranial Injuries	*George W. Swift*	510
Craniocerebral Trauma	*K. Winfield Ney*	520
Fractures of the Lower Jaw	*Robert P. Bay* *Brice M. Dorsey*	532
Prevention and Correction of Facial Disfigurement	*Vilray P. Blair* *James Barrett Brown* *Louis T. Byars*	536
Abdominal Catastrophes Resulting from External Violence	*Arthur M. Shipley* *John C. Hamrick*	542
Traumatic Appendicitis	*Henry H. Kessler*	555
Examination of the Injured Back	*John D. Ellis*	561
Effect of Trauma on Mechanically Weak Back	*Thomas P. Goodwyn*	577
Diagnosis and Treatment of Acute and Chronic Back Pain Due to Trauma	*C. R. G. Forrester*	582
Trauma of the Coccyx and Coccygodynia	*Monte Edwards*	591
A Plea for Conservatism in Fusing Operations on the Spine and Pelvis	*G. W. N. Eggers*	595
Rupture of the Supraspinatus	*Ernest A. Codman*	603
Compound Fractures of the Elbow Joint in Adults	*William R. Cubbins* *James J. Callahan* *Carlo S. Scuderi*	627
Injuries to the Carpal Bones. With Particular Reference to the Carpal Scaphoid	*Francis B. MacMillan*	633
Injuries to the Hand	*Torr Wagner Harmer*	638
Functioning False Joint of Finger Following Severe Trauma	*James L. Mangano*	659
Fractures of the Upper Extremity of the Femur—Neck and Trochanter	*W. Rogers Brewster*	662
Internal Derangement of the Knee Joint	*George E. Bennett*	670
Stabilization of the Ankle Joint Following Malunited Fractures	*Frank Goodwin*	679
Fracture of the Os Calcis	*Paul B. Magnuson* *Frank Stinchfield*	685
Compression Sprain of the Foot	*Fred M. Miller* *Thomas C. Browning*	693
Embryologic and Anatomic Considerations in Etiology of Inguinal and Femoral Hernias	*Leigh F. Watson*	695
Injection Versus Surgery in the Cure of Hernia	*Frederick W. Slobe*	704
Repair of Severed Tendons	*Leo Mayer*	714
A New Concept of Tenosynovitis and the Pathology of Physiologic Effort	*Nelson J. Howard*	723

Diseases of the Peripheral Arteries and Their Reaction to Trauma	*Bertram M. Bernheim*	731
Peripheral Nerve Injuries and Fractures	*Walter G. Stuck*	735
Treatment of Industrial Burns	*Herbert Poyner*	744
A Consideration of Some Traumatic Eye Conditions and Their Treatment	*Clyde A. Clapp*	750
Visual Acuity, Its Relation to the Form Sense and the Application of This Relationship to Medicolegal Problems	*Albert C. Snell*	756
Diagnosis and Treatment of Ruptured Urethra	*Amos F. Hutchins*	765
Effect of Trauma as "Precipitating" Factor in Certain Neurologic Diseases	*Foster Kennedy*	769
Functional Disorders of Nervous System as Sequelae to Trauma	*A. M. Ornsteen*	772
Roentgenologic Considerations of Certain Joint Injuries	*Carlton F. Potter*	785
X-Ray Examination of Clavicles and Acromioclavicular Joints	*Edward C. Holmblad*	791
The X-Ray in the Treatment of Infection	*Albert Soiland*	798
Aggravation of Chronic Pulmonary Tuberculosis by Trauma of the Chest	*J. Burns Amberson, Jr.*	804
Gas Gangrene. An Analysis of 109 Cases Encountered in Civil Practice	*C. Latimer Callander*, *Arthur Haim*, *Alexis Maximov*	811
Sulfanilamide in Surgical Practice	*Harold R. Bohlman*	824
Disability Evaluation	*Earl D. McBride*	840
Short Wave Diathermy in Industrial Rehabilitation	*Frank H. Krusen*	845

NEW SERIES, VOL. XLII OCTOBER, 1938 No. 1

The American Journal of Surgery

is the leading independent surgical Journal. It publishes many papers read before the outstanding Surgical Societies, but it is *not* "the official organ" of any organization.

Copyright, 1938, by The American Journal of Surgery, Inc.

EDITORIAL BOARD

Editor:

Thurston Scott Welton

New York

D. C. Balfour *Rochester, Minn.*
Carl Beck *Chicago*
George R. Brighton *New York*
Meredith F. Campbell *New York*
James T. Case *Chicago*
Isidore Cohn *New Orleans*
Bradley L. Coley *New York*
F. A. Coller *Ann Arbor*
George W. Crile *Cleveland*
E. C. Cutler *Boston*
R. V. Day *Los Angeles*
Charles A. Elsberg *New York*
Herbert C. Fett *Brooklyn*
C. R. G. Forrester *Chicago*
H. Dawson Furniss *New York*
J. H. Gibbon *Philadelphia*
Emil Goetsch *Brooklyn*
Charles A. Gordon *Brooklyn*
Donald Guthrie *Sayre, Pa.*
A. E. Hertzler *Kansas City*
Louis J. Hirschman *Detroit*
James M. Hitzrot *New York*
Frederick C. Holden *New York*
Emile F. Holman *San Francisco*
J. A. Hyams *New York*
R. H. Jackson *Madison*
John E. Jennings *Brooklyn*
W. L. Keller *Washington, D.C.*
H. A. Kelly *Baltimore*
T. J. Kirwin *New York*
Arthur Krida *New York*
A. V. S. Lambert *New York*
Maurice Lenz *New York*
H. H. M. Lyle *New York*
Jerome M. Lynch *New York*
Urban Maes *New Orleans*
Harrison S. Martland *Newark*
Rudolph Matas *New Orleans*
Roy D. McClure *Detroit*
H. C. Naffziger *San Francisco*
C. R. O'Crowley *Newark*
Louis E. Phaneuf *Boston*
Eugene H. Pool *New York*
Douglas Quick *New York*
N. P. Rathbun *Brooklyn*
H. A. Royster *Raleigh*
Henry S. Ruth *Philadelphia*
A. C. Scott *Temple*
M. G. Seelig *St. Louis*
J. Bentley Squier *New York*
H. J. Stander *New York*
George W. Swift *Seattle*
H. L. Updegraff *Los Angeles*
Grant E. Ward *Baltimore*
J. H. Woolsey *San Francisco*

EDITORIALS

INTRODUCTORY NOTE

PERHAPS it is a trite expression to say that, "All of surgery is not the aseptic scalpel." Obvious as such a statement is, many of our younger men fail to appreciate the major part which preoperative study and postoperative care play in the final result.

Perhaps preoperative study plays a greater part in ophthalmic surgery than it does in general surgery because it not only includes data from a general history and physical examination, but also that from an ophthalmic history and examination. The modern means available for ophthalmic study include microscopy of the living eye, and also studies which but a few years ago were found only in the physiologic laboratory. The old axiom, "To know, one must measure," can be applied in a practical way to nearly all of our methods of ophthalmic review.

Ophthalmic postoperative care is a science in itself. The inflammatory response of the non-vascular transparent tissues of the eye is not paralleled by any other structures, and it is often years before the beginner learns to interpret the changes met with. One must learn to distinguish those features characteristic of the disease for which the operation is performed, from those changes incident to the traumatism of the procedure.

The local effect of the various medicaments must be thoroughly understood, and sharply defined changes are often recognized from a single application of some agent.

It is not uncommon to hear interns and observers remark that so-and-so is a "good surgeon." They little realize that they are judging merely by his surgical dexterity. A man of much less digital skill may be a far superior surgeon, judging by better results due to the general management of his cases.

In reading the papers of this symposium, the reader should keep in mind not only that the views and opinions of the various contributors may disagree, but also that no attempt was made to supply a continuous or complete review of modern surgical ophthalmology.

John N. Evans, M.D.

EDEMA

One of the body's defense mechanisms which would seem to be a paradox is edema. Any injury to living tissue brings about a sudden and transient constriction of the capillaries which is very quickly followed by a dilatation. This dilation results in an increase in the permeability of the capillary wall and the tissues are therefore flooded with fluid from the blood stream. Aside from diluting the irritating substance in the tissues this edema interferes with the usual forces available for combating the irritant. The circulation is impaired, thereby decreasing the efficiency of cellular response. Antibodies, as well as the irritating substance, are diluted. In the case of infection in the nose and sinuses there is stagnation of secretions and an increased opportunity for the multiplication of organisms. There is also a decrease in aeration of the cavities and folds of the nose which probably plays an important part in infection. The ostia of the sinuses become blocked.

The only means for the escape of this accumulation of fluid is by way of the local system of lymphatics leading to the deeper lymphatic beds. If the source of the fluid is stopped the accumulation quickly drains away, allowing the tissues to return to their normal thickness and density. If the lymphatics become blocked this cannot take place.

If the edema persists and is profuse there develop herniations of the mucous membrane, these being the polyps and polypoid nodules so frequently seen in chronic infection and allergy. When this occurs the elasticity of the fibrous and elastic tissues has been lost and the resulting polyp remains until removed, due to the inability of the tissue to squeeze the fluid out through the lymphatics.

To combat an acute cold or sinusitis we are in the habit of keeping the nasal mucosa thinned out as well as possible without needlessly traumatizing and causing further irritation to the tissues. Vasoconstrictors decrease the source of edematous fluid, giving the excess a chance to drain off. This effect is only too temporary but seems to be of value. Whether we arrived at this method of treatment empirically or through such reasoning I do not know.

In the allergic mucosa, edema soon affords a foothold for any organisms entering the nose or sinuses. As long as the edematous state persists surgery as a means of eradicating the infection is futile. The opened antrum or exenterated ethmoid again becomes lined with a lifeless boggy tissue thrown up into folds which continue to harbor the organisms and stagnant secretions. The edematous condition of the tissues must be relieved before surgery is undertaken.

Perhaps one exception is in the case of bacterial allergy where the elimination of foods and inhalants under suspicion gives no relief. If a killed suspension of the organism cultured from the nose gives a strongly positive skin reaction, bacterial allergy should be suspected and the infection drained with autogenous vaccine therapy as an additional measure if the case tends to be stubborn.

Localized collections of edema are often controlled by the use of escharotics, cautery or ionization. Whatever is used the end result is the same, namely, the formation of a scar extending from the epithelial layer to the turbinate bone which splints the mucosa in such a manner that it is unable to swell.

In non-allergic vasomotor rhinitis the capillary dilatation and edema may result

not from a local irritation, but from an irritation of the skin, the intestinal tract or some other remote area. Vasomotor rhinitis of this type seems best treated by a routine of exercise, fresh air, good elimination and a good diet, factors which improve the tone of the smooth muscle of the vessel walls. A sharp septal spur, however, may be a source of mechanical irritation in the nose and its removal may give complete relief.

There is another form of edema of the nasal mucous membrane which is due to a different mechanism—the edema seen in patients suffering from dietary inadequacy. This type of edema is due to a disturbed albumin-globulin ratio in the blood plasma, resulting in changes in osmotic pressure. It may be a generalized edema of the whole body. The nasal mucosa is identical in appearance with the allergic mucosa, but eosinophils are absent from the nasal secretion. This of course responds rapidly to correction of the diet.

It will be seen that if the fundamental pathology is to be corrected in many of the above conditions our methods of treatment must be conservative. Trauma only increases the existing edema. It is true that cases of purulent sinusitis will come to surgery eventually in most instances but all of them should have the benefit of dietary correction and allergic study first if either is indicated.

L. W. Dean, Jr.

Subscribers to The American Journal of Surgery *visiting New York City are invited to make the office of the publishers (The American Journal of Surgery, Inc., 49 West 45th Street, New York) their headquarters. Mail, packages or bundles may be addressed in our care. Hotel reservations will gladly be made for those advising us in advance; kindly notify us in detail as to requirements and prices.*

EYE, EAR, NOSE AND THROAT SYMPOSIUM

Eye Section

EDITED BY

JOHN N. EVANS, M.D.
Professor of Ophthalmology, Long Island College of Medicine, Brooklyn, New York

THE USE OF THE ORBICULARIS PALPEBRARUM MUSCLE IN SURGERY OF THE EYELIDS

JOHN M. WHEELER, M.D.*

Professor of Ophthalmology, College of Physicians and Surgeons, Columbia University

NEW YORK CITY

THE orbicularis of the eyelids can be used to advantage in the correction of defects in the region of the outer canthus and in pronounced ptosis and in spastic entropion of the lower eyelid. I should like to speak briefly of methods of use of the orbicularis as applied to these conditions.

FIG. 1. Congenital anomalies of both eyelids. Absence of the outer canthal ligaments and failure in the formation of outer canthi have resulted in peculiar deformities.

Figure 1 illustrates a case of *congenital absence of the external canthal ligament*, with deformity of the outer canthus. Orbicularis strips were used to act as external canthal ligament and to hold the outer ends of the tarsal plates together, so that an outer canthus would result. An incision was made in the outer part of the upper lid about 3 mm. from the margin. A similar incision was made in the lower lid. Strips of orbicularis were dissected up and left attached near the outer ends of the tarsal plates. (Fig. 2.) The skin was undermined and the periosteum of the outer orbital margin was exposed. The flaps of orbicularis were crossed and attached to the periosteum of the zygomatic bone. (Fig. 3.) A gut suture was carried through the tissue at the outer end of each tarsal plate and tied so that a new canthus was formed. After suture of the eyelids the palpebral fissure was approximately normal in length and it was well shaped. The same procedure was performed on each side and the patient was enabled to open and close the eyes naturally. After operation there was hardly any appreciable deformity on either side.

In pronounced *ptosis of the upper eyelid* the best result is obtained by attachment of the lid to the superior rectus muscle or the eyeball. No other principle is acceptable if the eyelid makes no attempt to follow the eye in upward gaze. Various schemes for attachment have been suggested, but probably the orbicularis muscle furnishes as satisfactory material for suspension of the lid as can be obtained. In using strips of orbicularis of the upper eyelid, no deformity results from the lid dissection

* Deceased, August, 1938.

and there is no injury of the superior rectus. A skin incision is made a little above the upper border of the tarsus where the normal crease in the skin of the upper lid is placed. The skin below the incision is dissected from the orbicularis muscle and strips of muscle tissue about 4 mm. wide and 10 mm. long are dissected up, and attachment is left toward the middle of the eyelid. These attached ends are about 8 mm. apart and between them the orbicularis is left undisturbed. Above the tarsus, dissection is carried through the tarso-orbital fascia and the levator palpebrae superioris. The superior rectus is dissected out and a probe passed under cover of the muscle. During this dissection the conjunctiva of the upper fornix is pulled well down out of place and held down by a suture, so that it is not injured when the superior rectus is exposed. The strips of orbicularis are attached to the upper surface of the superior rectus by fine cat gut sutures and the skin wound is closed. After this procedure the eyelid follows the eyeball in upward gaze and a good palpebral fissure results. Temporarily, the eyes remain slightly open in sleep, but there is never dangerous exposure of the cornea as a result of operation. Figure 4 shows the attachment of the strips of orbicularis to the superior rectus of the eyeball.

Fig. 2. A horizontal incision has been made in the region of the outer canthus. Strips of orbicularis muscle have been dissected and left attached at their outer ends.

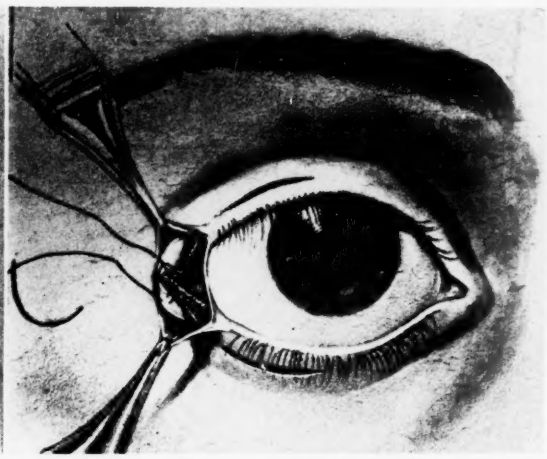


Fig. 3. The skin has been dissected from the underlying tissue, and the free ends of the orbicularis strips have been crossed and attached to the periosteum of the zygomatic bone.

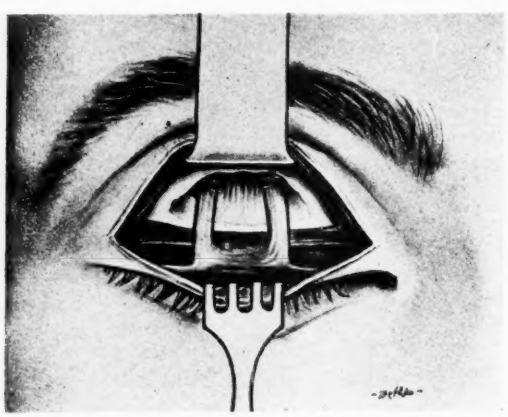


Fig. 4. Ptosis correction by attachment of orbicularis strips to the superior rectus. Dissection has been carried through skin, orbicularis, tarso-orbital fascia and levator palpebrae to expose the superior rectus. The orbicularis strips are attached to the superior rectus by fine gut sutures.

A *spasmodic inversion of the lower eyelid* can be corrected by several procedures which pull down the lid. In an attempt to find an operation that would correct the inversion of the lid and at the same time support it, I turned to the use of strips of orbicularis. Figure 5 shows a strip of the muscle attached to the periosteum of the zygomatic bone. A skin incision is carried along the outer two-thirds of the eyelid into the malar region. In direction it follows the contour of the lower eyelid and it is placed

about 5 mm. from the lid margin. A strip of orbicularis 4 mm. wide is dissected from the outer two-thirds of the eyelid a little below the tarsus. It is then attached to the periosteum of the malar bone and made quite taut. This strip does not laterally displace the lid margin because it is taken from a place below the tarsus rather than from one attached to it. The tautness of the strip holds the lower border of the tarsus back against the eyeball and prevents inversion of the lid margin. At the same time it gives good support to the lid.

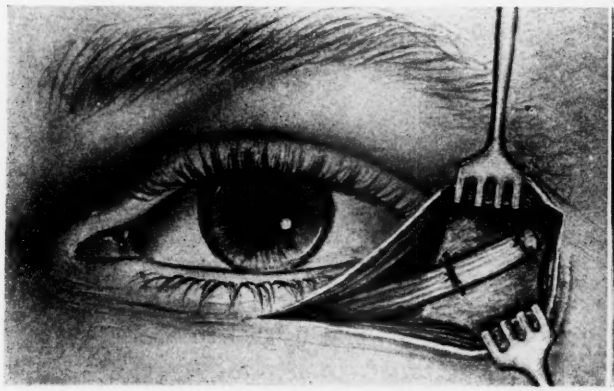


Fig. 5. Correction of spastic entropion by attachment of a strip of orbicularis to the periosteum of the zygomatic bone. This maneuver prevents inversion of the lid margin and gives the lid proper support.

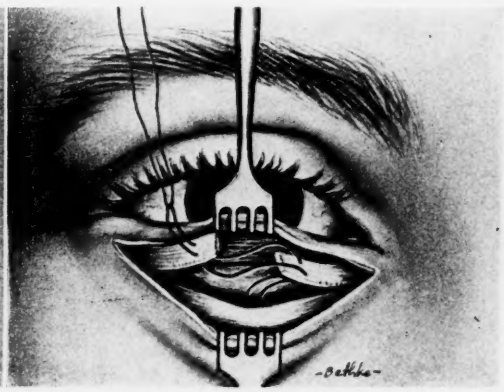


Fig. 6. Correction of spastic entropion of the lower eyelid by overlapping of strips of orbicularis and attachment of the overlapped strips to the tarso-orbital fascia just below the tarsus.

Figure 6 illustrates a slightly different use of orbicularis strips for the same purpose. An incision 5 mm. from the lid margin is carried the length of the palpebral fissure. Incisions through the orbicularis are made about 4 mm. apart to include that portion which overlies the lower part of the tarsus. The dissected strip is cut in the middle and the attachments near the outer and inner canthi are left undisturbed. A fine, double-armed gut suture is carried through the tarso-orbital fascia below the tarsal plate. Then both needles are carried through the strips of orbicularis 4 to 5 mm. from their ends. When the suture is tied an overlapping of 4 or 5 mm. is accomplished and the orbicularis is thrown in contact with the tarso-orbital fascia just below the tarsus. Pressure of the shortened strips at the lower tarsal border prevents entropion, without carrying the lid down out of place. This operation is effective and easily accomplished. If the skin incision is carefully closed the skin scar is not obvious.

In dissections of orbicularis muscle strips, infiltration of the tissue is a help, as the increased bulk that the infiltration gives makes an accurate dissection easier than it is in un-infiltrated tissue. The operations here described can all be done under local anesthesia, and novocaine infiltration is satisfactory.

It is entirely likely that other uses for the orbicularis muscle in surgery of the eyelids will be devised and I hope that the procedures briefly described here will serve as fair examples of effective, simple operations.

SURGERY OF SECONDARY GLAUCOMA

ALLEN GREENWOOD, M.D.

Professor Emeritus of Ophthalmology, Tufts College Medical School

BOSTON, MASSACHUSETTS

IT is at once apparent in considering this subject that one must give some mention to other than surgical procedures, such as those which precede the actual surgery. Ophthalmic surgeons are always, in all cases of ocular disease, on the lookout for that justly dreaded complication, secondary glaucoma.

It is well to keep in mind some of the physical differences between secondary glaucoma and the primary types. The main difference is that in secondary glaucoma the increased intraocular pressure is due to changes in the fluids of the eye which hinder absorption, and the presence in the fluid of cellular elements and other materials which are deposited in the angle of the anterior chamber and block the passage.

Some types of secondary glaucoma are especially disastrous, as for example that which frequently follows a complete obstruction of the central vein and occasionally follows a partial obstruction. For this reason, I have made it a rule to prescribe pilocarpine for all patients with central vein obstruction in hopes of avoiding glaucoma. Sometimes I have thought I had achieved this result. Complete obstruction of the central vein always produces an altered condition of the intraocular fluids, and frequently this alteration shows itself in an acute iritis, which is quickly followed by a secondary glaucoma. Whether an iritis always precedes a secondary glaucoma in these cases it is often hard to decide, since the two conditions come so close together.

Given a complete obstruction that results in a blind eye followed later by sudden hypertension, there is only one adequate surgical measure, i.e., enucleation. Attempts to lower the tension by miotics, iridectomy, trephine, iridencleisis-sclerectomy, sclerotomy, or other measures are disappointing, and enucleation is the final resort.

In the presence of a fulminating glaucoma where the previous history is unknown or not suggestive of central vein thrombosis, the use of miotics gives valuable information in that if, under pilocarpine and eserine, there is no lessening of the pain, no contraction of the pupil, and no improvement in the vision, one naturally suspects the type of glaucoma formerly spoken of as hemorrhagic glaucoma. In most cases of acute congestive glaucoma not of secondary origin, such medical treatment usually shows some improvement. In partial obstruction of the central vein or one of its main branches, where fair vision is left, a secondary glaucoma may be precipitated. In such cases it is sometimes possible permanently to cure the glaucoma by some form of filtrating cicatrix, especially if there is a lessening of tension and an improvement in the eye condition following the use of miotics. Recently I saw a young woman who was sent to me for enucleation of an eye which had a secondary glaucoma following a partial central vein obstruction. She had some vision left, and the use of miotics and adrenalin improved the vision and the condition of the eye so much that I did a combination iridencleisis and sclerectomy. Today this young woman has a vision of $^{20}/_{40}$ in an eye that I was asked to enucleate. For certain of my cases of chronic glaucoma I have found the above mentioned operation extremely useful. I have, in times past, advocated this special type of procedure and think it wise at this time very briefly to describe the operation and its advantages.

The operation consists in laying down a conjunctival flap similar to that for the

trephine operation, but without splitting any of the layers of the cornea. The incision is made as far back in the angle of the anterior chamber as possible and in pulling the conjunctival flap forward a good beveled lip of sclera is easily demonstrated. A portion of this lip may be cut off with the Lagrange scissors or, as I prefer it, punched out with the scleral punch. Usually one bite of the scleral lip with the punch is sufficient, but sometimes one or two extra ones are made each side of the central one. The iris is then pulled out and the sphincter cut on one side of the forceps hold, or sometimes on both sides. This latter makes a tongue of iris tissue with the black pigment surface facing upward. The conjunctival flap is then replaced and fastened with a running suture. The great advantage of this operation over the simple iridencleisis is due to the fact that no massage is ever necessary in order to establish the filtrating cicatrix. A conjunctival flap wide enough to cover the tongue of iris is essential.

Another form of secondary glaucoma which is not amenable to any of the ordinary surgical procedures is that which comes in the late stages of a sarcoma of the choroid. Usually a diagnosis of sarcoma is made long before the advent of the glaucoma; but I have had one case of fulminating glaucoma with the cornea so steamy that a view of the fundus could not be obtained, where an iridectomy was performed and immediately on the clearing up of the eye a sarcoma became visible and an enucleation imperative. Such cases, however, must be fairly infrequent, though occasionally reported.

Another type of secondary glaucoma that is very difficult to handle by surgical measures is that which occurs in tuberculous uveitis. In such cases if all non-surgical measures have proven useless, then repeated paracentesis is the safest surgical procedure and is often sufficient. It has recently been demonstrated that in tuberculous uveitis that a paracentesis or repeated paracenteses will render the eyes more amenable to treatment with tuberculin on account of the greatly increased number of antibodies thrown into the aqueous after a paracentesis. I feel that this simple procedure for bettering the condition of the aqueous is not taken advantage of nearly so frequently as it should be. It removes an unhealthy aqueous and allows it to be replaced by a fresh and more normal fluid with its increase of antibodies, and also reduces tension.

Over twenty years ago I began treating all patients with ocular tuberculosis with tuberculin. I quickly arrived at the conclusion that the long continued use of minimal doses was the proper way to use tuberculin. I have had many patients who have had the tuberculin injections over a period of many years, more or less continuously, taking care never to increase the doses to the point of causing focal reaction. This method of desensitizing the patient has certainly given me excellent results. The more radical surgical procedures are very apt to be followed by disastrous results besides proving ineffective. Some of these cases will, in spite of everything, result in enucleation.

The glaucoma that sometimes results from a contusion of the eye, with or without partial dislocation of the lens, is usually amenable to medical treatment. Where this is unsuccessful, permanent help is obtained by a simple iridectomy.

In any case of acute iritis, especially where there is a good deal of foreign material deposited in the anterior chamber, one must be on the lookout for secondary glaucoma. When it occurs, instead of using a miotic, one frequently gets better results by pushing the atropine to the limit with the addition of some form of adrenalin, such as adrenalin bitartrate, and then using measures to relieve the ocular congestion. I have had some very excellent results from the use of sulfanilamide, particularly in the iritis of sympathetic and late infection. Good results also may be obtained from the use of one or two leeches applied to the temple about 1 inch back of

the external canthus, in addition to pushing the atropine. If the glaucoma does not subside under this treatment, repeated paracenteses may be sufficient, particularly if combined with the customary use of typhoid vaccine as a foreign protein. If this mild surgical measure is not sufficient, an iridectomy will almost always accomplish the desired result. In all cases of secondary glaucoma that come with iritis or uveitis one may keep in mind the fact that the secondary glaucoma does not quickly produce disturbance in the optic nerve or the visual fields. I have seen a secondary glaucoma last a good while and then on its cure have had the vision return to normal. Also, I have never seen a deep, undermining cupping of the optic nerve in cases of secondary glaucoma.

The secondary glaucoma that sometimes comes from a lenticular intumescence where a cataract is beginning, does not yield to local treatment but requires some form of surgical interference. Some surgeons in such cases advise a combined extraction of the lens in order to relieve the patient of both the cataract and the glaucoma at the same time. Personally, I prefer to do the iridectomy first and then extract the lens later. In such cases one must try, if possible, to differentiate between the secondary glaucoma that has come from the swollen lens from a concurrent chronic glaucoma and beginning cataract. The possibility of a chronic glaucoma supervening on a beginning cataract provides one reason why I like to see my cataract patients fairly frequently.

Sometimes when a cataract follows a perforating injury to the anterior portion of the eyeball, a glaucoma will be precipitated and require immediate attention. The best surgical practice is that of immediate removal by linear extraction of the swollen lens substance, and thorough irrigation or suction removal of any remaining lens cortical. Personally, I have found suction much more efficacious than simple irrigation. The same treatment holds good for the glaucoma that sometimes complicates a needling for congenital cataract. Fortunately, I have never seen this complication, as it has been my custom in all cases of needling for congenital cataract thoroughly to open the anterior capsule so that any swollen lens substance will be easily poured into the anterior chamber where it quickly absorbs.

Following a cataract extraction a glaucoma will sometimes develop, particularly if there are any tags of capsule or iris left in the wound, not properly covered by conjunctiva. In many of these cases, treatment with miotics over a long period will take care of the secondary glaucoma. If this does not come about, a surgical procedure is necessary. The cyclodialysis has been favored by some surgeons. I have had good results from a trephining, from an iridencleisis, or from a sclerectomy. In cases where it becomes necessary to operate on the capsular cataract which sometimes follows an extracapsular operation, the needling will often allow sufficient vitreous to come into the anterior chamber to start up a secondary glaucoma. This may be remedied by miotics or by some form of operative interference; I have found that a good iridectomy has worked out very well. If this is unsuccessful, then some filtrating operation or a cyclodialysis may be used. Along this line should be mentioned those cases where an extraction for senile cataract is done by the method of Homer Smith, consisting of capsulotomy the day before the actual removal of the lens. In some of these cases a glaucoma will quickly result and be immediately relieved on completion of the operation.

Sometimes a shrunken lens will be freed from its zonular attachment and become dislocated into the anterior chamber, inducing a glaucoma almost immediately. It is possible in some cases to massage the eye while the patient is lying down, and the lens may slip back into the posterior chamber. I feel, however, that it is much better immediately to extract the lens, always using a miotic to shut down the

pupil and make a broader band of iris to protect the lens from slipping into the posterior chamber during the attempts at extraction. Some operators have extracted such lenses by having the patient operated on with his face turned downward. I have never found this necessary, since, with the pupil somewhat contracted by the miotic, I have had little difficulty in removing the lens.

Each case of secondary glaucoma must, of course, be treated according to the primary causes. Whatever surgical measures are used must be selected for the needs of the individual case, keeping in mind all the different factors involved.

Virchow is popularly known as the father of pathology. His claim to paternity may be legitimately extended to include modern medicine, for during the activity of Virchow, there was hardly a prominent medical worker in the world who had not studied under him, or whose activity had not been deeply affected by his contributions.
From—"The Postmortem Examination" by Sidney Farber (Thomas).

THE TECHNIQUE OF EXTRACTION OF INTRAOCULAR FOREIGN BODIES

WALTER B. LANCASTER, M.D., F.A.C.S.

Ophthalmologist, Massachusetts General Hospital; Consulting Surgeon in Ophthalmology, Massachusetts Eye and Ear Infirmary

BOSTON, MASSACHUSETTS

THERE are two divisions of the subject, extraction of (1) magnetic and (2) non-magnetic foreign bodies.

Three techniques are used for magnetic extraction:

1. The old diascleral method: incision with one thrust through all the coats of the eye into the vitreous at the point of of election (down and out), insertion of the magnet pole into the vitreous, probing if necessary.

2. The anterior method: a giant magnet gradually approaching the center of the cornea, the foreign body is pulled forward through the vitreous around the lens into the posterior chamber, and thence extracted through the pupil and cornea, preferably with a hand magnet.

3. The modern diascleral method embodying various improvements not described by Wagenmann in his 1870 page monograph in the Graefe Saemisch Hess Handbuch nor by Haab in his 125 page monograph in the same Handbuch: exact localization of the foreign body either by *x*-ray or by application of the hand magnet to the sclera, incision by layers with diathermy knife to avoid hemorrhage, applying tip of hand magnet to incision, extraction without entering vitreous, placing a ring of diathermy micropunctures around the incision to forestall separation of the retina.

Certain considerations are common to all these methods and will therefore be discussed first.

Any intraocular foreign body is a menace. Even the blandest materials such as gold or smooth glass, cause an inflammatory reaction, which *may* go on to fatal damage to the eye. In many cases, however, a tiny sterile fragment becomes encapsulated and may remain a long time without serious harm. Hence every case presents a problem. Can this particular foreign body be left to nature? Is the harm sure to be done by trying to extract the foreign body likely to be greater than would result from expectant treatment? For it must be remembered that just as the foreign body causes damage on its way into the eye so it will cause damage (often much more) on its way out.

Delay may make removal far more difficult. It may be that the patient is seen so soon after the injury that the foreign body is visible in the fundus. In a few hours the lens may become so cloudy that no view of the fundus is possible, thus making the extraction far more difficult if the foreign body is non-magnetic. If the foreign body is not sterile or has carried in germs from the conjunctiva in passing, a few hours may result in infection. For example, Wagenmann reports a case in which the patient, a medical student, presented himself within an hour after the injury. The foreign body, steel, had entered the eyeball but a little point projected far enough to be grasped with forceps so that the foreign body was extracted and immediately cultured. There was copious growth of cocci. The eye healed promptly with good vision. How long could the foreign body have been left in the tissues without setting up infection? The best rule is: Take no chances, but remove at the earliest possible moment, not waiting for an *x*-ray unless there is some strong special reason for it.

If the fragment is sterile but consists of some irritating material, like copper, it *may* become encapsulated but the chance

of this is very small. The more likely course will be the formation of sterile pus and the gradual, not very slow, destruction of the eye.

Thus the *first principle* to be established is that the technique of extraction calls for the utmost effort to save time. Get the foreign body out while the getting is good: before the lens becomes opaque, infection is established or chemical irritation has set in.

The *second principle* is this: Weigh the chances of a successful attempt at extraction against the chance that nature will take care of it if left alone. Give due consideration to your own experience, the equipment you have available, the results you have had in the past. You may feel that it will be better for the patient if you do not operate. Perhaps another surgeon would be more likely to succeed. The patient should be given the opportunity to avail himself of an operator with better equipment and a good record in such cases. I have known of patients sent by airplane over a thousand miles for this reason—and with successful results. The question of time must be taken into account. Wise and conscientious decision may be and often is extremely difficult, especially in a one-eyed patient.

These principles apply to both magnetic and non-magnetic foreign bodies. Another principle of universal application is the necessity of most careful and detailed study of how the accident happened. The posture of the patient at the time, the direction of his gaze, the manipulations that were going on, usually hammering of some sort, how the tools were held, inspection of the tools, the site of the wound of entrance, the pathway of the foreign body inside the eye, the ophthalmoscopic examination with well-dilated pupil, and lastly, but often of prime importance, an accurate radiologic localization.

One of the first questions to be decided may be whether to wait until a radiologic examination and localization are accomplished. Many surgeons argue that an operation for extraction of a foreign body is so important and serious that it should not be undertaken until all available data have been collected. The fact is, however, that 80 per cent of the small magnetic foreign bodies in the eye behind the lens can be extracted just as well without previous localization provided the anterior method is used. To postpone for a few hours may be a serious mistake. If the anterior method fails or if the foreign body is suspected of being too large for its safe use, then an *x*-ray may be taken, unless the operator decides that it is safer to proceed at once to try a diascleral operation. This will depend on whether he can locate the foreign body with the magnet. If not, then by all means an *x*-ray localization is demanded.

Other matters common to the different techniques are:

Careful cleansing of the eye, removal of dirt, removal of prolapsed tissue, rarely replacement of clean, freshly prolapsed tissue, removal of tags and crushed tissue, coaptation of lips of wound by suture or more often by conjunctival flap to cover wound. If vitreous threatens to prolapse through the scleral wound, paracentesis of the anterior chamber, by reducing tension, acts favorably during the operation and so does good deep anesthesia.

If the patient is first seen several days after the accident, one may freshen the edges of the wound (perhaps with the cautery) before flapping. If the lens is swollen and increased tension calls for it, linear extraction may be performed, or paracentesis may be done and repeated. If paracentesis is done by cautery it will drain for several days. In threatening infection great stress was formerly laid on mercury, then on salicylates, later on vaccines, and now on foreign proteins intravenously (especially typhoid vaccine). Even if hypopyon is present, the eye is not necessarily doomed. Removal of the foreign body and vigorous treatment may save the eyeball.

It may be that the patient is sure that there is no foreign body in the eye—

only a cut. However, one should take no chances, but have an *x*-ray at once, unless the giant magnet is used for diagnosis and proves positive. Many tragic cases could be cited to bear out this advice.

Susceptibility of the eye to foreign body injury is influenced by its previous condition. Myopia and poor vision from any cause tend to cause the worker to be nearer his work and so more likely to be injured. Myopia and vitreous and retinal conditions which favor separation of the retina; chronic inflammatory conditions which threaten to flare up; glaucoma; degenerated vessels; weak lens support, either congenital or acquired, which favors subluxation of the lens; general constitutional diseases such as renal, vascular, syphilitic, diabetic and anemic; focal infections; local conditions which favor infection, such as dacryocystitis, conjunctivitis and blepharitis; general and hygienic conditions under which the patient lives and works, heat, dust, sweat—all these have a bearing not only on prognosis but on treatment.

A foreign body in the eye does damage: (a) mechanically, direct and indirect, (b) chemically, and (c) by infection. One must decide what share each plays in the picture.

Anesthesia is an important part of the technique. In children and some adults narcosis is necessary; in most cases local anesthesia supplemented by a liberal dose of nembutal, sodium amytal or the like, is satisfactory. If the giant magnet is to be used for diagnosis, only superficial anesthesia is needed since the patient's sensation of pain is an important guide. If the diagnosis is already established as to the presence of a foreign body, it is better to have good deep anesthesia (orbital injection of procaine plus suprarenin) before applying the giant magnet, because the pain may be severe when the foreign body hits the iris from behind.

A powerful hand lamp to illuminate the field of operation is indispensable and a binocular loupe is important for some of the operative procedures, such as detecting slight movements under the influence of the magnet.

EXTRACTION OF MAGNETIC FOREIGN BODIES

Although in extracting non-magnetic foreign bodies, invisible in the fundus, accurate radiologic localization is indispensable, this is not true with magnetic bodies. Of course accurate localization is an advantage, but one must weigh the advantages of time-saving in the other scale of the balance. If the case is fresh and the foreign body has not had time to become entangled in fibrin and beginning encapsulation; if it is not embedded in tissue, but only enveloped in vitreous with perhaps a little blood clot, *it is not necessary to know the exact localization.*

The proper technique will depend on the size of the fragment, the amount of damage done on its way into the eye, the site of the wound of entrance and on the quality of the equipment available, together with the experience of the operator and his knowledge of how to use magnets.

Suppose the surgeon concludes from the wound of entrance, the appearance under focal illumination (if possible with the slit-lamp), and under ophthalmoscopic examinations, after giving due weight to the circumstances under which the injury occurred, that the foreign body is small and is in the posterior part of the eye—exact site unknown. If he has a powerful giant magnet, he can adopt the following technique: The eye having been prepared for operation by cocainizing and irrigation, the patient is seated before the magnet and a very good light thrown on the eye. The surgeon stands in front and to one side of the patient. His hands steady the patient's head and are so disposed that they rest against the magnet pole and against the patient's face, bridging the space between. They are not behind the patient's head pushing him forward but in front holding him away from the magnet. The patient is told to push for-

ward against the surgeon's hands, being assured that the surgeon will not permit the eye to bump against the magnet. Should the patient feel pain or alarm, there is nothing to prevent his pulling back. Finding himself free to move except forward, he is not afraid and pushes gradually forward. The surgeon manipulates the head so that the eye gradually comes to touch the tip of the magnet. No current is on. This is preliminary to gain the patient's confidence.

Now the head is held off a distance of 6 to 8 inches and the foot switch closed by the operator. At this distance, the pull is very feeble on a small fragment and probably nothing will happen. The patient does not feel anything, the surgeon does not see anything. The foot switch is released. The head is moved a few inches nearer and the current switched on again. The head is so held that the axis of the magnet corresponds with the visual axis of the eye. This requires some object for the patient to look at in order that the eye may be held steady. A small flashlight held by an assistant so that the patient can fix it with his other eye will suffice. Sometimes one may just tell the patient to stare straight ahead. The foot switch should be closed only a few seconds at a time. Opening and closing several times at short intervals (1 or 2 seconds) helps loosen the foreign body. If nothing happens, the eye is made to approach nearer and nearer until there is contact between the cornea and the tip. If there is no result, the eye is held at a short distance from the tip and the patient is made to look in different directions by placing the flashlight in suitable positions. The axis of the magnet should be made to coincide with a line from the center of rotation of the eye to the margin of the cornea, now in one meridian, now in another. The foot switch is closed and opened two or three times in each new position. The idea is to extricate, if possible, by pulling in different directions the foreign body which is not quite free to move.

When this technique is successful, the foreign body is pulled through the vitreous (always end foremost, never flat side foremost), until it impinges on the very convex and very smooth posterior surface of the lens. Almost invariably, the foreign body glides around the lens still pointing toward the point of the magnet. It now passes through the fragile zonule of the lens and impinges against the iris. The surgeon (who has been watching intently for this) sees this as a bulging of the iris and the patient feels it as pain. The patient draws back and the surgeon releases the foot switch simultaneously.

The foreign body is now in the posterior chamber. It is possible in many cases to manipulate the eye and the giant magnet so as to pull it through the previously dilated pupil into the anterior chamber and after a suitable corneal incision pull it out. This is distinctly bad technique. The proper procedure is to place the patient on the operating table and proceed to extract the foreign body with a small hand magnet which can be manipulated with ease and precision (for details, see below). Some have advocated using a polar extension of some sort; a probe or nail or other instrument, one end of which is in contact with the magnet and so becomes magnetized, using this to extract the foreign body. This too is very bad technique. Sometimes it can be made to work. I can imagine doing a cataract operation with a pocket knife and no other instruments but what a poor substitute for proper equipment and approved technique! If the giant magnet fails to pull the fragment around the lens even after considerable coaxing, then two alternatives present themselves.

1. The giant magnet may be applied to the point of election back of the ciliary body, say at a point between the lateral and the inferior rectus. It may be that being somewhat nearer the foreign body and pulling at a different angle it will succeed in pulling it to this point of election. The evidence will be the pain

felt and *the slight bulging of the sclera* at the point where the magnet pulls the foreign body against the wall of the eye. An incision may be made in the sclera at this point (according to the technique given below), and the foreign body extracted with a small magnet. An alternative method is to reapply the giant magnet at the center of the cornea when probably the now loosened foreign body will be pulled around the lens in the usual way. One has pulled the fragment in a zigzag path, first from its original site to a point of election down and out, then by another step forward around the lens. Obviously, more damage has been done than by the preferred method of pulling straight forward through the vitreous, but much less than by probing through the sclera with a small magnet.

2. The other alternative is to treat the patient as a subject for diascleral extraction and follow the technique for that method.

3. Sometimes the wound of entrance is behind the cornea and it is possible by applying the magnet at the wound of entrance to pull the fragment out the way it went in. If this procedure is chosen, it is well to apply the *small* magnet first at the wound of entrance using the bluntest tip which has most action at a distance. If this should pull the foreign body to the wound it is done with less trauma than would be caused by the clumsy giant magnet.

It is sometimes argued that it invites considerable damage to the eye to pull the fragment forward, unless it is very small. Suppose the surgeon concludes, after weighing the facts obtained by studying the case, that it is not advisable to attempt extraction by the anterior method. The foreign body may be so large that to pull it around the lens would be likely to damage the lens. If the lens has already been damaged by the foreign body on its way into the eye, as happens in 50 per cent of the cases, this argument fails. The risk of pulling it forward is the risk of dragging a rather large foreign body through the vitreous and so around the lens where it may scrape the ciliary body and then bump into the iris. To this must be added possible damage to the retina (if the foreign body was embedded in it) when it is pulled away.

Some of these injuries are inevitable in the nature of the case. If the foreign body is embedded in the retina, it must be pulled out if it is to be removed from the eye. It must be dragged through the vitreous, but that is less harmful than it would be to insert a small magnet tip into the vitreous and pull the fragment out that way. Damage to the ciliary body might be caused if the eye were not in proper position with its visual axis parallel to the axis of the magnet. If clumsily used, the magnet would possibly pull the fragment against and then drag it along the ciliary body into the posterior chamber. Then it would be possible to cause an iridodialysis by grossly overdoing the pulling. In other words, most of the damage to be feared by pulling the fragment forward around the lens is either damage that is unavoidable, if the foreign body is to be removed at all, or damage that is due to unskillful use of a powerful instrument.

The hand magnet has too small a field to pull fragments at a distance. If no giant magnet is available or if it has failed, then the diascleral method must be used. Since the hand magnet will not pull a fragment from a distance, it must be brought into actual contact or within a millimeter or two of the foreign body. This means that the foreign body must be accurately localized. Then an incision is made directly over the foreign body and it is extracted.

To localize the foreign body, chief reliance must be placed on the *x*-ray. One can be aided by observing the path followed by the foreign body on entering the eye, which may be evident from the positions of the wounds in the cornea, iris, lens, and vitreous. If seen very early, the foreign body may be visible in the fundus. If the retina has been hit and damaged, a scotoma may locate it exactly.

There is another aid to localization not generally utilized but which I have often found valuable. When one has a general idea of about where the foreign body is, the problem is to locate it so exactly as to be able *to cut down directly onto it.* The tip of the small magnet is applied tentatively and systematically over the suspected area. The tip is pressed lightly against the sclera at right angles and lifted a couple of millimeters then applied again and lifted. When the application is made directly over the foreign body (which is separated from the magnet only by the thickness of the coat of the eyeball), there is a slight bulging, a slight sensation of attraction which shows that the foreign body is just underneath. If a fragment of steel, say a short piece of a broken needle, is placed under several layers of paper or a layer of towelling and the operator is not told just where it is, he can readily locate it by this method. This will serve to show him how to make the manipulations on the eye.

The foreign body is located after the sclera is exposed by a suitable conjunctival flap, and an incision must then be made into the vitreous. This was formerly done by single thrust of a Graefe knife, making a meridional incision either curved (because a curved incision gapes easier), or L-shaped or T-shaped. This not rarely caused a hemorrhage into the vitreous. It is still advised by Comberg in Berens' textbook. By using a diathermy knife or needle with a cutting current, the incision may be made without danger of hemorrhage. Care should be taken, however, not to allow the current to act on the vitreous.

When the tip of the magnet is applied to the incision—not introduced into the vitreous—the fragment is so near and the modern hand magnet is so strong that usually the fragment is felt at once to be drawn to the tip. If it is brushed off by the lips of the wound the lips may be retracted by non-magnetic hooks. The incision in the sclera may need a suture, depending on its length and its tendency to gape.

Before closing the conjunctival flap, one should make a ring of micropunctures with a diathermy needle and coagulating current in order to set up adhesions between the retina and choroid to forestall separation of the retina.

This technique is far superior to the old method of cutting boldly through the sclera, choroid and retina into the vitreous, then inserting the pole as far as necessary to reach the foreign body, or if its exact location is not known probing around till it is found. The *immediate* results of these old diascleral operations were good. The long range results were not. Separation of the retina, hemorrhage in the vitreous, vitreous opacities, and shrinking of the vitreous were common later consequences. The late results after extraction by the anterior method were far better.

Magnetic Foreign Bodies Anterior to the Vitreous. 1. *Magnetic Foreign Bodies in the Lens.* The authorities with the largest experience and study of the literature (Wagenmann, Haab, Elschnig, etc.), advise immediate extraction of the foreign body with the giant magnet. If the patient is seen early, it is often possible to pull the fragment out the way it went in. If it has been long in the lens, especially if it is small, it may not yield to the pull of the magnet. The capsule wound may have closed. A radial puncture of the capsule and lens as well as the cornea with a Graefe knife affords an opening through which the fragment can be pulled with a giant magnet, or even sometimes with a large hand magnet. If the pupil is quickly contracted with eserine, the iris will block the opening in the capsule and allow the capsule to heal rapidly, sometimes avoiding a total cataract. If one waits until the lens has become opaque, one has to operate more blindly.

The old technique was to wait until the lens became opaque and then extract lens and foreign body at the same time. It is not a sure way, since the foreign body may have become adherent to the posterior capsule and, having rusted, may be hardly magnetic and difficult to get out. If the

patient is old enough for an intracapsular extraction, it may be wise to wait until the capsule is healed and then extract lens and foreign body at once.

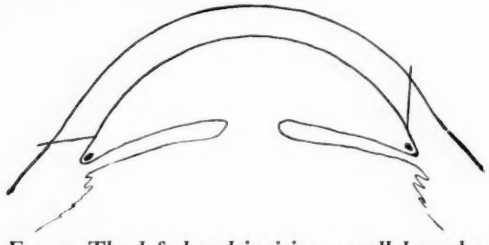


FIG. 1. The left hand incision parallel to the plane of the iris in the usual way is incorrect. The foreign body cannot be reached with forceps or hook if non-magnetic, and is not so easily extracted even with a magnet as when the incision is made, as on the right side, perpendicular to the plane of the iris, allowing easy access to the angle.

2. *Magnetic Foreign Bodies in Posterior Chamber.* This is commonly a stage in the extraction of deeply seated foreign bodies by the anterior method. The giant magnet should not be used for this stage of the operation, since the objects can be coaxed into the anterior chamber through the dilated pupil much more safely with the hand magnet. The things to avoid are (1) injury to the anterior lens capsule and (2) excessive injury to the iris. It is best to hold the magnet so that the pull is oblique between iris and lens, remembering that the foreign body always moves so that its long axis is parallel to the pull of the magnet, i.e., it always comes end on, never sideways.

3. *Foreign Bodies in the Anterior Chamber.* When the foreign body is in the anterior chamber, it often falls to the bottom and may be out of sight, but is easily pulled by the hand magnet. In making an incision into the anterior chamber for the purpose of extracting a foreign body, either magnetic or still more if non-magnetic, the incision should not be made as most corneal incisions are made—parallel to the iris. This makes the peripheral lip overhang the angle in such a way that an instrument, magnet tip if the foreign body is magnetic, or hook or forceps if the foreign body is non-magnetic, cannot be inserted in the direction of the angle of the chamber. It would be necessary to go around over the edge of the wound to get to the angle.

The correct technique is as follows: The foreign body is usually below. Make puncture and counter-puncture at or posterior to the limbus with a Graefe knife, at 5 and 7 or even nearer 6 o'clock, with the edge of the knife upward away from the limbus and cutting forward and upward, making a small curved flap ending about 2 to 4 mm. from the limbus, depending on how large an incision is needed. The bevel on this is such that one has easy access to the angle with any instrument. The bevel is approximately at right angles to the plane of the iris instead of parallel to it. The size of the incision should be adapted to the case. If forceps are to be used (non-magnetic foreign body) a larger flap than for a magnetic foreign body is needed. The pupil should be contracted strongly. Once more good light and magnification are very important, as is good anesthesia. If the iris should be pulled into the wound, it should be replaced, not iridectomized.

NON-MAGNETIC FOREIGN BODIES

A non-magnetic foreign body in the cornea projecting into the anterior chamber may present a difficult situation if the foreign body is so far in that attempts to dig it out with a needle will probably push it into the anterior chamber. Two techniques have been developed for this: (1) A bent broad needle with a blade 4 or 5 mm. wide is entered obliquely through the cornea and across the anterior chamber close behind the foreign body and making counter-puncture beyond. This will support the foreign body and prevent its escaping into the anterior chamber while it is being dug out. (2) A large corneal flap is cut, large enough to be everted so that the foreign body can be grasped with forceps on the inside and pulled through.

Any manipulation which permits the point of the foreign body to hit the capsule is highly objectionable.

Non-Magnetic Foreign Bodies in the Anterior Chamber. The incision for these is the one described above, made by cutting away from the limbus instead of towards it as usual. A blunt hook or forceps of suitable design can be inserted into the angle. The pupil should be contracted. A good light is indispensable. An iridectomy should be avoided. The corneal incision may be closed with a conjunctival flap.

Non-Magnetic Foreign Bodies in the Posterior Chamber. These are rare. The foreign body should be located by *x*-ray if sufficiently opaque. The pupil is dilated with euphthalmine and adrenalin. Then with a blunt hook or suitable forceps, one can fish for the foreign body behind the iris, taking care not to injure the lens. After its extraction, the pupil is contracted to avoid anterior synechia.

Non-Magnetic Foreign Bodies in the Lens. Such objects may be eased into the anterior chamber with a needle, working through the cornea, and then extracted through the proper corneal incision. The lens may be extracted and, with good luck, the foreign body with it. Sometimes a small foreign body heals in the lens and causes no trouble, at least for many years.

Non-Magnetic Foreign Bodies in the Vitreous or Retina. Successful extraction requires exact localization. If seen before the lens is opaque so that the foreign body can be seen through the dilated pupil, manipulations can be directed by sight. The instruments, hook or forceps of suitable design, are introduced through a window in the sclera and guided by observation through the pupil. The ocular endoscope of Thorpe[1] is designed to favor manipulation in the vitreous by a similar optical arrangement to that in the urethroscope or nasopharyngoscope, etc.

When the foreign body is in the wall of the eye and is accurately localized by *x*-ray, one can (often enough to keep up his courage for the next case) cut down directly onto the foreign body and extricate it safely.

For the most part, non-magnetic foreign bodies in the posterior part of the eye offer insuperable difficulties which challenge the ingenuity and skill as well as the courage of the boldest operator.

CAUSES OF FAILURE OF OPERATIONS FOR REMOVAL OF FORIEGN BODIES FROM THE EYE

1. The foreign body has caused extensive damage to the eye because of its large size: hemorrhage, loss of vitreous and of other contents of the eye.
2. Infection introduced by the foreign body or by the operation.
3. Delay in getting under treatment.
4. Difficulty in locating the foreign body was formerly a cause but not now, except in the case of certain non-magnetic foreign bodies which do not show in the *x*-ray.
5. Foreign bodies embedded in tissue. This will prevent extraction by the anterior method, but with good localization the surgeon should be able to apply the small magnet to the sclera over any desired spot. This will often require temporary resection of one of the rectus muscles to provide access to the eyeball posterior to the equator. This is much safer than probing from in front through an incision in the sclera down and out.
6. The most frequent cause of failure in attempting to extract magnetic foreign bodies is adequate equipment.

In extraction by the anterior method, a very powerful giant magnet, if used early, will succeed in pulling all but the very smallest fragments which are enveloped in clot or fibrin or in tissue. Haab estimated that 80 per cent, and Comberg that 70 to 80 per cent of the foreign bodies in the posterior part of the eye can be dealt with by the giant magnet, leaving 20 per cent to 30 per cent which must be approached diasclerally. Obviously, the figures will depend on the equipment as well as on the skill of the operator.

ADEQUATE EQUIPMENT

A giant magnet should be tested by its power to pull a small fragment (say a steel ball 1 or 2 mm. in diameter—not 4 mm. in diameter as advised by Volkmann), at a distance of 20 mm. from its tip. A good magnet will pull with a force fifty to eighty times the weight of the fragment at this distance.

A small magnet to be manipulated with one hand cannot in the nature of the case have useful power at a distance of 20 mm., but in contact it should have a very powerful pull 4,000 to 8,000 times the weight of the test ball, which should be of the smallest size. It should have a strong pull at 2 mm. distance, which is about the farthest it has to pull in practice. The hand magnet pictured by McDannald[2] probably would not pull one-hundredth part as much even with direct current and not one-thousandth part as much with alternating current as a "good" hand magnet. The permanent magnet of hardened steel may be expected at its best to pull about one-hundredth part as much as a "good" hand magnet.

Compare this power of a good hand magnet with that of a rod, chain, probe, or the like, with one end on a giant magnet and the other end by induction acting as the magnet. In this case the pull will be less than one-hundredth as much as the hand magnet and in addition manipulation is incomparably less easy and precise than with the hand magnet. Only the profoundest ignorance of how magnets work could lead one to endorse this, as Haab and others have done.

A foot switch controlled by the operator himself is indispensable. The flash from the switch which opens and closes the current to the giant magnet is so large that a remote control switch is desirable. This can be located in any convenient part of the building away from the operating room or in a box. It is also highly desirable with very powerful magnets to have a surge control to take care of the tremendous voltage of this spark which may break through the insulation of the magnet coil and seriously damage it. Haab, Volkmann, Schlosser and even Hartmann use only moderate power, less than a kilowatt, and so the flash is much less and the danger also less. The Lancaster giant magnet, using 3 to 10 kilowatts, is really in a class by itself.

A good light concentrated on the patient's eye is essential.

A rheostat is superfluous except that the hand magnet may not be made to carry the full voltage of the building without a resistance to control the current.

An ammeter is not essential, but is useful.

Much has been written about the design of eye magnets, and a large part of it is not worth the paper on which it is written. This is deplorable since the fundamental principles of electromagnetism were worked out in the last century. Such principles as are important for the eye surgeon are set forth in my paper, written in 1915.[3]

For a given weight of iron and of copper wire, the best form of magnet is a cylindrical core about 1 meter long and 12 cm. in diameter, ending in a bullet shaped tip and wound with a coil extending as far as possible toward the end of the core, i.e., to the point where it begins to taper. The larger the wire the less the resistance and the more amperes will go through it. The magnetizing power depends on the number of amperes multiplied by the number of turns of wire around the core. For a given length (or weight) of wire, there will be more turns if it is wound along the whole length of the core than if it is heaped into a large coil at one end. There will be more amperes the larger the wire. Hence the rule is to use as large a wire as the supply of current will permit. For a giant magnet, 20 amperes is the least, 50 amperes is better. It might seem that many turns of small wire with a smaller current would give the same result as fewer turns with a larger current, but that is not true. About this there is no uncertainty.

It has been stated that the magnetizing force depends on the ampere-turns. This

takes no account of the core. The core is used because it very greatly increases the magnetism resulting from the magnetizing force. A coil with no core or a wooden core would give a certain amount of magnetic power, as does for example, the *innenpol* magnet of Mellinger. But if a core is inserted inside the coil, it enormously increases the magnetism, not simply 100 per cent but many thousand per cent. If the core is small, it helps, while if it fills the whole coil, it helps much more. Yet recently Mellinger innenpol magnets were made standard for all the big machine shops in Russia! I have met English and American surgeons who believe that this most faulty design is a good one. *Testing the pull on a small steel ball at 20 mm. is the final criterion. Theories which do not meet this test must be scrapped.* Unless a giant magnet will pull a tiny steel ball with a force over fifty times its weight at a distance of 20 mm., it is not a "good" giant magnet. Unless a hand magnet will pull a tiny steel ball in contact with its tip with a force over 5,000 times its weight, it is not a "good" hand magnet.

A magnet induces magnetism in any iron or steel in its field (for example a watch in the pocket if one is standing within a meter or so of a giant magnet). When magnetized, a bit of iron has a north and a south pole, no matter how small it is. What most writers have not understood is that the north pole of the magnet *repels* the north pole of the fragment of iron and *attracts* the south pole. Hence if the field were of uniform intensity, the repulsion of one pole would exactly equal the attraction of the other. It is solely due to the fact that the intensity of the field diminishes as one gets farther from the pole of the magnet that the pull on the nearer pole of the fragment is greater than the repulsion of the pole that is farther from the magnet, and so the fragment as a whole is attracted to the magnet. Hence the absurdity of Mellinger's claim that the uniform field of the ring magnet is the basis of its superiority. The field is not uniform and if it were there would be no pull!

Another important, very important, consequence of the attraction of one pole and repulsion of the other is that the fragment always moves toward the magnet end on, never side on. Also a fragment which is long and slender, like a bit of broken needle, is pulled more forcibly per unit of weight than a chunky fragment, for instance a sphere. The reason is that there is a greater difference between the intensity of the fields at the two ends in a longish fragment, with the pull at one end so much more powerful than the repulsion at the other.

The monographs by Wagenmann and by Haab in the Graefe, Saemisch, Hess Handbuch, Vol. IX, are very exhaustive. They cover diagnosis, prognosis, treatment, etc., very fully and critically, and I have derived much valuable material from them. Unfortunately, they do not give reliable critical evaluation of the many forms of electromagnets which they describe. They do not claim to be experts in that subject and depend on engineers or physicists who ought to know, but do not always. Their bibliographies are exhaustive. Haab says his engineers do not agree with all that I claim in my paper,[3] although no one has called in question or disputed any single statement, much less disproved it.

Comberg, in Berens' textbook,[4] states that it is sometimes necessary to apply the giant magnet to the eye every day for a week "in order to magnetize the foreign body." This shows profound ignorance of the principles of magnetism, since the foreign body is magnetized with the speed of light, practically instantaneously.

SUMMARY

Lose no time as infection may have time to set in or fragment may become embedded.

X-ray is not needed for magnetic foreign bodies in the majority of cases.

(*a*) 70 to 80 per cent can be pulled forward from the posterior part of the eye, through the vitreous, around the lens, into

the posterior chamber by a good giant magnet.

(*b*) If imbedded in the wall of the eye, this is impossible. Then localization is essential. There are three important means: (1) careful study of the path of the foreign body; (2) *x*-rays, which give pretty close localization but not so precise that one can be sure to cut down exactly on the foreign body; (3) for exact final localization, a short tip of a good hand magnet applied to the sclera tentatively and systematically over the suspected area. The tip is pressed lightly on the sclera and then lifted a couple of millimeters and again applied and lifted. The slight bulging of the sclera and the slight sensation of attraction reveal the exact site of the foreign body. Access to the posterior part of the eye is possible, as in operations for separated retina, by temporary section of a rectus muscle and the use of a good retractor (non-magnetic). Cutting through the wall of the eye by the thrust of a Graefe knife may cause damaging hemorrhage. To avoid this use a diathermy knife. It is not necessary to probe the vitreous with the magnet or even to penetrate the vitreous if the foreign body has been precisely localized. Application to the lips of the wound suffices if the magnet is a good one. Avoid a giant magnet for foreign bodies that are within 2 or 3 mm. of the magnet. Avoid extensions applied to the giant magnet to act as hand magnets.

After extracting diasclerally, apply a ring of diathermy micro-punctures around the site of the scleral wound to forestall separation of the retina.

Results, especially late or end results, of extraction by the anterior route with the giant magnet are much better than final results with diascleral extraction by the old method, still widely used. Statistics are not available for the more modern technique here described.

Details of technique for foreign bodies, magnetic and non-magnetic in different parts of the eye—cornea, anterior chamber, posterior chamber, lens, vitreous, imbedded in the wall of the eye. Importance of good light.

Six causes of failure: Most important is inadequate equipment. What constitutes adequate equipment and how to test it. Hand magnet in contact should pull a fragment well over 5,000 times its weight. Giant magnet at 20 mm. should pull a fragment well over fifty times its weight. Obviously, a magnet pulling fifty will fail oftener than one pulling eighty.

REFERENCES

1. Thorpe, H. E. Ocular endoscope. *Tr. Sect. Opbtb., A.M.A.*, pp. 290–291, 1934.
2. Berens, Conrad. The Eye and Its Diseases, p. 1028. Phila., 1936. Saunders.
3. Lancaster, W. B. Stronger eye magnets. *Tr. Am. Opbtb. Soc.*, 1915.
4. Berens, Conrad. Op. cit., p. 1128.

TECHNIQUE OF SCLERAL FIXATION OF EXTRAOCULAR MUSCLES

P. CHALMERS JAMESON

Senior Surgeon, Brooklyn Eye and Ear Hospital

BROOKLYN, NEW YORK

THAT definite scleral suturing was not practiced until the last decade as a general accompaniment of muscle on the recessed side seems strange. It was, however, probably due to lack of a technique which could confute the frequently quoted statement as to the danger of the scleral suture.

Dr. Edward Jackson[1] in 1903 reported a case of operation on the tendon of the superior rectus muscle for paresis of the superior oblique in which he sclerally fixed the muscle. Dr. R. I. McCurdy[2] reported a recession of the rectus in conjunction with advancement in which he sutured to the superficial sclera. Dr. Claude Worth,[3] when he published his advancement operation, described his technique for suturing to the sclerocorneal margin.

Not until 1922, when I reported the first fifty cases of single recession[4] ever recorded to my knowledge, was a definite technique described for safe scleral suturing. I realized that if the entity were to succeed, scleral suturing, especially in the region of the equator and the region posterior to it, must be demonstrated as safe and reliable. The episclera had been cited as the only safe means of fixing the muscle, but the episclera is defined in Gould as "the loose connective between the conjunctiva and sclera," and this is not reliable material for anchorage. The sclera proper, as experimentation of the human (enucleated) eye demonstrated, is extraordinarily strong. Even a superficial bundle of fibers can hold much weight. A suture was introduced into the sclera and tied, and to this silk suture a weight of 1½ pounds (608 Gm.) was attached before it tore out. Moreover, the sclera, when the eye was later opened, was found to be intact and unbroken on the inner surface. The strength of the scleral fiber is almost inconceivable.

The technique of scleral suturing should be mastered before practicing recession. If one can secure an enucleated eye, it makes good material to practice on. The method I devised is based upon the very apparent observation, that a thin lamella of scleral fiber is semitransparent, and a needle passing underneath such a surface layer is visible in its bend, as also the suture when drawn is visible. Contrariwise, the bend of a needle which has perforated and takes in the whole thickness of the sclera is invisible and cannot be seen, nor can the suture when it is drawn.

I can do no better than to quote from my previous report on the subject:[4]

"The field and sclera should be sponged of all débris and exposed smooth and white as marble. The needle must be sharp, small, strong and slender, full curved 14 mm. from tip to eye. A large blunt needle will burst and mutilate the fibers and will cramp the operator for room. The needle engages carefully but boldly at first and the point always watched and visible is driven through translucent sclera, picking up bundle after bundle of fibers. As the point becomes indistinct it is made to approach the surface but not put through the surface (until exit of needlepoint), steady pressure being exerted as it makes its way through the tissue and engages in its bend substantial bundles of fibers. The motion employed is such as one could adopt in picking up the fibers of a heavy linen handkerchief bent over the finger without pricking the underlying finger. The bend of the needle after counter puncture should

be visible in its entire continuity through the translucent sclera as also the suture after it is drawn."

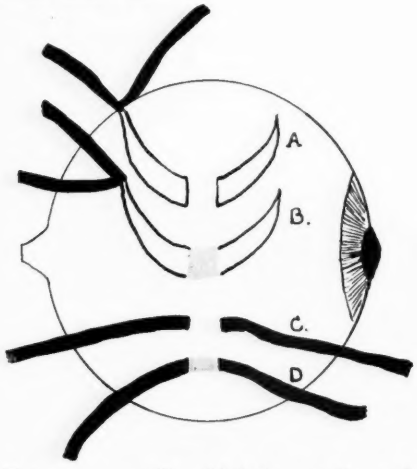


FIG. 1. A, needle which has perforated sclera. B, needle which has not perforated sclera. C, suture of perforation. D, suture of non-perforation.

A caliper may be used to measure the gradation, but the experienced operator can estimate without it.

The above procedure provides a safe and practical technique in any portion of the sclera. Ten or twelve sutures on the enucleated eye following this outline will give the operator confidence, and when the eye is opened, non-perforation is revealed.

At this point in the evolution of the technique I had established the strength of the scleral fiber, and while the ability to sustain traction was amply demonstrated, there were other points to be considered.

Three regions on the sclera came under question: first, the most frequently used, the central region between the muscle insertion and the equator; second, the region behind the equator, less frequently used, but essential at times in cases of the giant congenital muscles, secondary contractions, strabismus fixus cases, and contraction syndrome cases; third, the region between the muscle insertion and sclerocorneal margin.

The region between the muscle and equator presented little difficulty. With its field always plainly visible, any small veins could be side-stepped and the technique above described could be practiced.

I was, however, summarily brought to a standstill when suturing behind the equator. The equator in relation to the muscle and cornea had not at that time been clearly defined. With early enthusiasm because of the results obtained by definite scleral fixation and gradation, I was not at first particularly exact as to the point at which the muscle was placed, and in two instances I went too far back (a mistake which cannot be made, as will be seen if Figure 2 is kept in mind), and was disappointed by my needle penetrating a punk-like area in which there was no scleral resistance. This was explained later, after the débris was cleared, and I realized that I had inadvertently penetrated the venae vorticosae, which in these cases were situated a little too far forward.

Figure 2 is self-explanatory, and helps to divide these two areas, as it delineates the equator. The left eye is represented and the space which theoretically can be recessed is delineated by the area which is lined in the illustration. The 5 mm., 4 mm., 3 mm., 2.5 mm. areas of recession corresponding to the internal rectus, inferior rectus, external rectus, and superior rectus are estimated on a small eye and are conservative; 1.5 mm. may be added to each in the average case.

The other portion of the sclera mentioned is that between the insertion of the muscle and the sclerocorneal margin, as used in the Worth and kindred advancements. There is some difference experienced in this region, in contrast to that of the equator. The sclera proper in this region is not, as generally supposed, very much thicker than at the equator. The general impression that it is thicker here is due to the fact that the capsule becomes intimately blended with it. I realized this when doing some experimental work on the vascularization of this part. The capsule cannot be curetted away from the sclera in this region, unless preceded by multiple sectioning. The Graffe knife, as it sections the capsular tissue, experiences only a cheese-like resistance, in contrast to the

resistance of sclera proper when reached. The separation is however, accomplished easily by the curette, when the sectioning is completed, and the sclera is left white and glistening as at the equator.

The suture then, if placed superficially in this region, *does not* reach true scleral tissue. I am guided in the depth I go more by the feel of the point of the needle than the bite in this superficial tissue, as there is marked resistance to the point of the needle when the sclera is reached which the experienced operator will sense. It is for the aforementioned reason that the bite taken by the needle, if superficial, is more likely to burst out and also why a deeper suture may be taken. The operator should, however, aim at including at least a superficial bundle of scleral fiber, and this, as stated, is obtained safely by tactile sensibility, but requires moderate experience.

Contrary to the general impression, suturing in this section is not dangerous. The writer has advanced in this region for forty years and has never had a serious reaction, much less the loss of an eye.

A further important consideration is the needle used in scleral suturing. The ordinary needles used in succulent tissues are not suitable for the sclera. The fibers are so compact and resistant to transit that, unless the blade of the needle is constituted to cut a channel which will admit of the free transit of the after coming heel with its suture content, the fibers are likely to be traumatized. A needle* I devised is shown in Figure 3; it presents a javelin-shaped blade, a tapering butt which is ingrooved

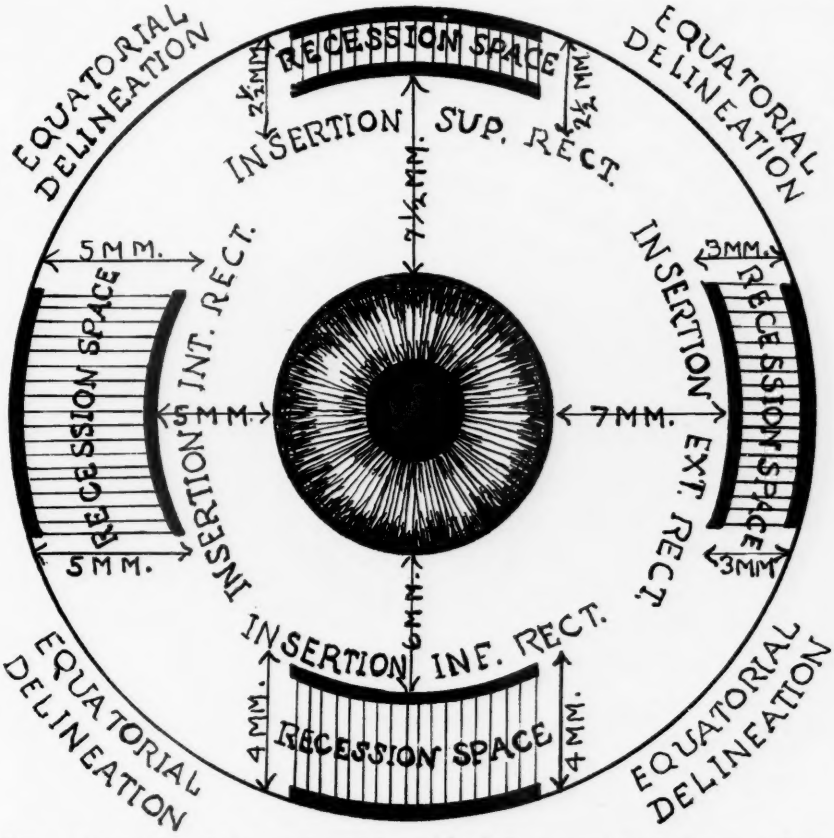


Fig. 2. Insertion of the recti muscles with their approximate distances from the sclerocorneal margin and approximate recession space from the insertion to the equator of the respective muscles.

* These needles can be obtained from P. Moria, 108 Boulevard St. Germain, Paris, France; E. B. Meyrowitz, 520—5th Ave., New York, or Paul Tiemann & Co., 107 East 28th St., New York.

to admit of the suture lying within the body.

I believe the suture material best

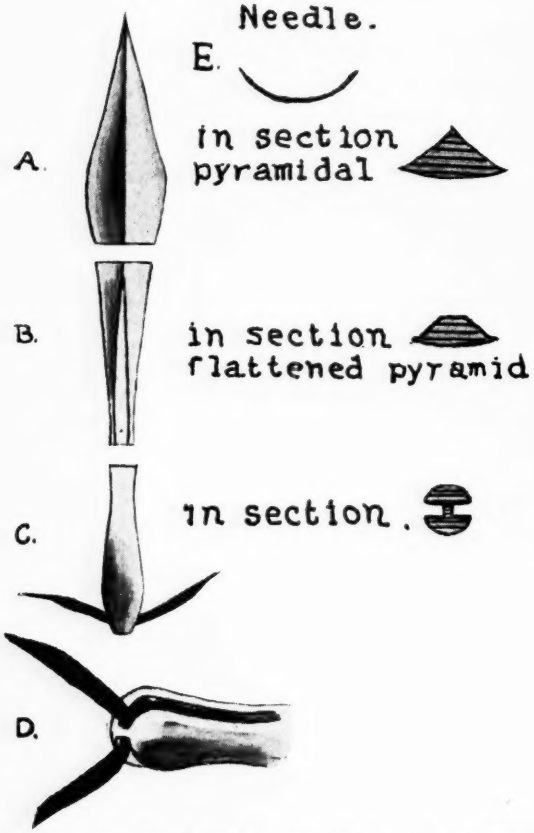


Fig. 3. A, javelin-shaped cutting blade. B, flat saddle so needle will not turn in holder. C, needle butt ingrooved on side. D, detail of butt and ingrooved eye. E, actual size of needle 14 mm. from eye to butt, full round.

adapted to scleral suturing is catgut. The No. 000 chromicized makes a good average strength. In contrast to silk, it has distinct advantages. Some of these I quote:[5]

"A silk suture becomes more irritating the longer it is retained. Catgut becomes less irritating the longer it is retained. Silk of small caliber tends to cut into tissue, especially if it is tied tightly and subjected to tension. On the contrary, catgut, by reason of increasing caliber, cuts less and holds without trauma. This principle is exercised by florists who use raffia in tying the stems of delicate cut flowers.

"Catgut, by its faculty of increasing caliber, occupies the channel made by the suture as its surrounding begins to fall away. Moreover, after it has served its purpose, catgut rapidly disintegrates. It lends itself to postoperative sterilization by its permeability and above all the suture does not require removal; for this reason, stitch abscesses from retained sutures, one of the causes of late infection after operation, is avoided and prevented. I allow the patient to be discharged from the hospital after the sixth day without removing the sutures, and have never experienced the slightest trouble."

If the catgut is not pliable enough when it is removed from its vial, it can be made so by placing it between the folds of a moist sterile towel.

The direction taken by the bite of the scleral suture is immaterial. It may be vertical or horizontal. Indeed, if in young subjects the operator finds it difficult to manipulate the needle on account of limited space, he may place the suture obliquely.

Scleral sutures should always be broadly placed both in relation to muscle and sclera. In the sclera they should be 3.5 mm. apart, and at least one suture should be secured in the muscle so that it will not pull out. In my recession work I half hitch the ingoing central muscle suture on the surface. At the final tying of this suture, which takes in conjunctiva, muscle and sclera, a bundle of muscle fibers is included, and these fibers can not pull out.[5]

In cases in which there is likely to be much tension, the ligated suture can be used.[6] The principle employed in this form of suture is that, after it is passed through either sclera or muscle, it is immediately tied. The bundles can not then pull out. The opposing sutures of muscle and sclera are then finally tied. This form of suture is the most secure, I believe, which can be practiced, and is therefore valuable when tension exists.

Finally, in safe scleral suturing there is the pathway to the development of muscle recession. It has therefore to a large extent revolutionized muscle work. For previously the surgeon could fix and grade on one side

alone; now he can fix and grade on both. He is enabled to estimate with much more accuracy the amount of correction required in a given type of case. He may determine the type of cases which should be recessed and those which should be advanced, and estimate in each instance how much may be accomplished by recession and how much by advancement; and finally, he can make his computations much more accurately, basing them on underlying conditions and type of muscle rather than by the estimation so long practiced of deviation alone.

SUMMARY

In this paper the subject of scleral fixation is thoroughly outlined and reviewed.

The strength of the scleral fiber is stressed. The technique of scleral suturing in the region of the equator is outlined and intimately described. The three regions in which scleral suturing can be practiced, i.e.: (a) behind the equator; (b) between the equator and muscle insertion; and (c) the region anterior to the muscle insertion, are discussed in detail. The instrumentation, especially the needle to be used is described. The advantages of catgut are cited. The ligated suture used in cases of tension is stressed.

REFERENCES

1. Jackson, Edward. Operation on the tendon of the superior rectus for paresis of the superior oblique. *Opbtb. Rev.*, 22: 61, 1903.
2. McCurdy, J. R. Operative treatment of squint. *Missouri State M. A.*, 13: 16, 1916.
3. Worth, Claude. Squint, Its Causes and Treatment. 5th Ed., p. 217. London, 1921. Balliere, Tindall & Cox.
4. Jameson, P. Chalmers. Correction of squint by muscle recession with scleral suturing. *Arch. Opbtb.*, 51: 5, 1922.
5. Jameson, P. C. Surgical entity of muscle recession. *Arch. Opbtb.*, 6: 329, 1931.
6. Jameson, P. C. Ligated suture. *Arch. Opbtb.*, 12: 377 1934.

PRESENT STATUS OF TENDON TRANSPLANTATION OF THE OCULAR MUSCLES*†

LUTHER C. PETER, M.D.

Professor of Ophthalmology, University of Pennsylvania Graduate School of Medicine

PHILADELPHIA, PENNSYLVANIA

DURING the last decade, surgery of the ocular muscles has undergone a refining process to meet the demands for greater precision, less trauma to the surrounding tissues and a minimum of postoperative reaction. The inspiration for refinement has its origin in the ability of the present-day surgeon to bring about binocular single vision with stereopsis in concomitant squint and in a sizable number of cases of paralytic squint.

The day of free and unrestrained tenotomies has passed, and this unsurgical procedure is replaced by tendon recessions and tendon advancements, selected according to the special needs of each patient. Whether the occasional operator, located in districts far removed from medical centers, can acquire the necessary delicate technique can be determined for himself by critical analysis of his surgical ability in relation to his patient's needs. In all probability, a conscientious surgeon may elect to limit his surgery to cases which call for simplified technique, and will refer his more complicated problems to consultants of wider experience.

In meeting the requirements of precision with a minimum degree of trauma and postoperative reaction, the question of anesthesia plays a major rôle. Many operators believe that local anesthesia is adequate, even in children of 12 years, but there are two objections to local anesthesia. In the first place, the operator is hindered in executing refined technique in most cases, and in the second place effective local anesthesia requires subconjunctival and intracapsular injections of cocaine, novocaine or some other drug, the immediate effect of which is the creation of edema with disturbance of the relations of muscle, capsule and conjunctiva. Remotely such injections bring about postoperative adhesions. In children under 12 years of age, gas-ether is the anesthetic of choice. In older children and adults, *avertin*, supplemented if necessary by vinethene, is the ideal anesthetic. If a more careful analysis were made of disappointing operative results, the character of the anesthesia would receive more attention.

RECESSIONS AND ADVANCEMENTS IN CONCOMITANT SQUINT

The most elementary forms of tendon transplantation to receive attention are recession to weaken the power of a muscle when it is indicated, and advancement to increase the power of a weak muscle. Which of these procedures is indicated in a given case is determined in convergent squint by a careful analysis of the degree of convergence for distance and the near point. If the squinting eye turns in excessively at the near point, a recession should be selected. If the squint is more pronounced at 6 meters an advancement may be indicated. In divergent squint a similar process of analysis will enable the operator to decide whether recession of an external rectus or an advancement of an internal rectus is the operation of choice. If the squint is not over 15 degrees and can be corrected by operation on one muscle, this method of reasoning should be the surgeon's guide, and not his preference for any single operation. True enough, instead of

* For brevity, the phrase "ocular muscles" will be used instead of the more accurate term "extraocular muscles."

† Most of the operations discussed in this paper may be found in detail in the author's book, "Extra-Ocular Muscles," Second Edition (Lea & Febiger).

advancing the external rectus, a tucking of the muscle may be adequate. The underlying principle is the important consideration. If the case is of such character that it becomes a surgeon's choice, advancement or shortening of an external rectus should be selected rather than a recession operation. Reasons for this plan of procedure will be set forth in subsequent paragraphs.

Recessions. In convergent squint, if dynamic convergence at the near point is excessive, a recession is indicated primarily. Squint of considerable degree may require a recession and an advancement. If an internal rectus is receded to a safe degree (not over 5 mm.) the most that can be expected in end results is a gain of 11 to 12 degrees. With good technique, a combination of a safe recession and an advancement may net as much as 25 degrees, although in most instances, the gain in correction may fall short of this amount from 5 to 7 degrees.

In the choice of technique, some operators will be partial to their own. Some may select that of Jameson, a detailed account of which may be found in Jameson's original paper[1] or as described by the author.[2]

The technique which the writer has practiced for many years and which he has recommended to the profession has certain advantages over the Jameson technique and will be given in detail. The advantages are: (1) simplicity of execution; (2) preservation of the greater part of the tendon (which is of paramount importance); and (3) a reduction in tissue destruction with less postoperative reaction and cicatricial tissue formation.

Independent of any technique which may be adopted, the operator should have regard (1) for accuracy in placing the scleral sutures so as not to cause subsequent hyper- or hypophoria; (2) to avoid residual cyclophoria; (3) to avoid convergence insufficiency by receding the muscle within safe limits (a recession of not more than 5 mm.); and (4) for undue destruction of the capsule of Tenon, which can be avoided by minimum dissection above and below the muscle and by blunt dissection of the capsule in the direction of the muscle fibers.

In the author's technique[2] the conjunctival incision is made to the outside of the plica semilunaris. A small opening is made into the capsule above the muscle, through which a muscle hook is passed to lift and separate the muscle from the surrounding capsule. The capsule is left intact above and below the muscle and its dissection is carried out, for the most part, in the direction of the muscle fibers. When the muscle and tendon are freed, a whip stitch suture of "000" ten-day catgut is introduced, as outlined in Figure 1.

The scleral bites are placed obliquely, care being observed that they are begun at the point from which the reattachment is to be placed. They are carefully centered so as to avoid reattachment too high or too low, and to avoid subsequent cyclophoria.

The sutures are passed through the tendon stump and tied at this point. The capsule and conjunctiva are closed with a running suture of No. 1 twisted silk (waxed).

Both eyes should be closed for four days. The conjunctival suture is removed on the fifth day.

In this operation, the tendon is preserved in its entirety and all sutures are placed in the tendon and not in the muscle. The capsule is disturbed but little and the reaction is negligible.

While the technique described, as a rule, is used on the internal and external recti, it is the best form of procedure when a recession of the superior or inferior rectus is indicated.

Advancements with and without Resections. Shortening of the external or internal rectus can be accomplished by tucking, by O'Connor's cinch operation, by resection and by advancement. This paper is concerned with tendon transplantation and the indications for the other shortening operations will not be discussed. Some consideration, however, should be given to

preference for an advancement over that of a simple resection. In the writer's experience nothing can be gained by a resection which tucking will not accomplish. When more effect is required than tucking can bring about, an advancement, with or without a resection, should be selected. Academic discussion as to whether an advanced tendon will become adherent to the old stump and exert its pull from this point, may be ignored.

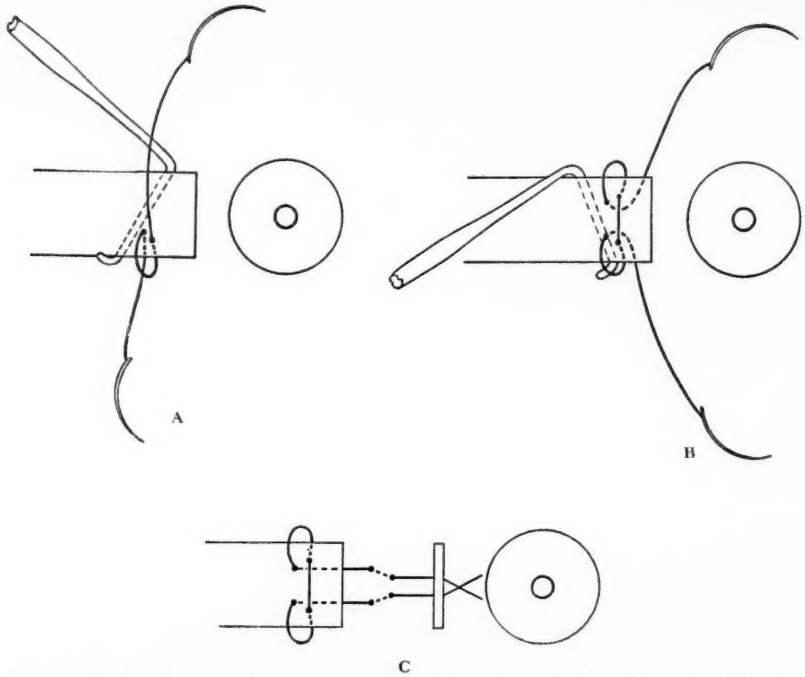


Fig. 1. Author's recession suture. A, introduction of the whip stitch in the lower third of the muscle by lifting the toe of the muscle hook. B, introduction of the upper stitch by lifting the heel of the hook. C, scleral bites introduced and the suture passed through the tendon stump and tied. (From Peter, "Extraocular Muscles," 2nd Ed., Lea & Febiger, 1936.)

The real indications for an advancement are: (1) to obtain a better fulcrum to increase power; (2) to limit the transplantation to the tendon and not to the muscle (which cannot be done in a resection); and (3) to furnish maximum results by advancing the tendon, capsule and conjunctiva. Operations which meet these objectives are the Worth advancement and that which the writer has devised and practiced for many years. The special indication for and the advantages of each will receive due consideration.

The Worth operation is the best technique ever proposed to advance the external rectus muscle in convergent concomitant squint. After the conjunctival incision is made, similar to that described for recessions, the muscle is exposed and elevated on a muscle hook. When the tendon is freed from Tenon's capsule, its own muscle sheath remaining intact, Worth's or Prince's or Jameson's advancement forceps is applied, an assistant or the operator drawing the conjunctiva and capsule forward beneath the outer or male blade. When the forcep blades are clamped, they will include the tendon, capsule and conjunctiva. The tendon is now detached, leaving a tendon stump of about 0.5 mm. The introduction of the sutures is followed easily by referring to Figure 2. After the sutures are introduced, the resected tendon, capsule and conjunctiva are cut back of the advancement forceps, and the sutures are tied. Both eyes are bandaged for five days and the sutures are removed on the seventh or eighth day.

The special advantages of this operation are: (1) it is unsurpassed in effectiveness by any other operation on the external rectus;

(2) the relation of the tendon, muscle and conjunctiva is disturbed but little; and (3) the postoperative reaction is slight.

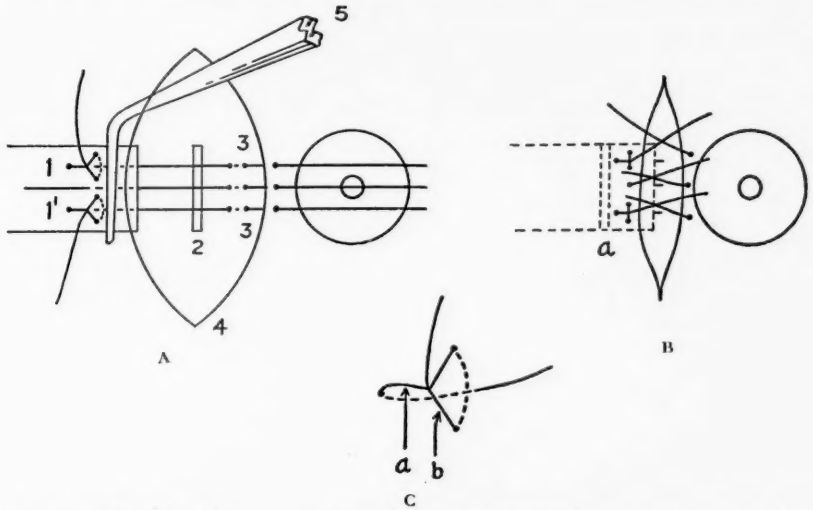


Fig. 2. Worth's advancement graphically illustrated. A, 1 and 1′ anchor sutures, above and below the guide suture, introduced through the conjunctiva, capsule and tendon. 2, stump of the old attachment. 3, scleral bites—the sutures finally emerging on the anterior surface of the conjunctiva. B, muscle detached and drawn into position and entire wound closed by tying of three sutures. C, *a* and *b*, points to be cut when anchor sutures are removed. (From Peter, "Extraocular Muscles," 2nd Ed., Lea & Febiger, 1936.)

The disadvantage of the operation is the slight difficulty in removing the sutures. The anchor sutures are cut at (a) and (b) in the illustration, Figure 2C.

The Author's Advancement Technique. When the internal, superior or inferior rectus muscle requires advancement, the technique which the author has devised and practiced will be found to be more satisfactory than that of Worth. It is equally applicable to the external rectus when it becomes necessary to advance and reattach it after a tenotomy which has been followed by postoperative convergent squint.

After the usual conjunctival incision is made, the muscle is freed by blunt dissection as described. A whip stitch of No. 3 twisted silk (waxed) is introduced as in a recession operation suture, and the muscle is severed close to its attachment. The conjunctiva and capsule are dissected from the sclera from the muscle stump to the limbus. The sutures are introduced as shown in Figure 3. The second scleral bite at (3) in this figure is reserved for a reattachment which may be required after tenotomy resulting in a divergent squint, and in cases in which the first scleral bite may be deemed inadequate. In the latter technique, the sutures should be drawn taut at (2) with Steven's forceps and finally

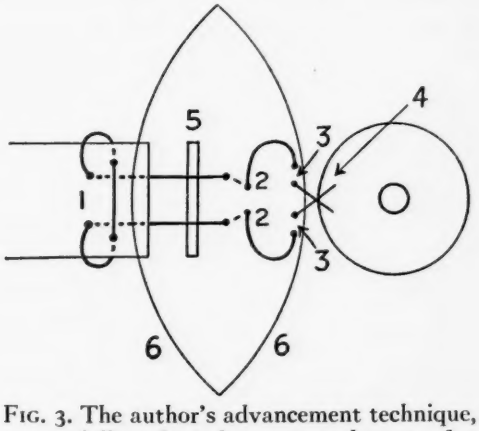


Fig. 3. The author's advancement technique, especially adapted to reattachment of a previously tenotomized internal rectus and to advancement of the inferior and superior recti. 1, whip stitch; 2, oblique scleral bites; 3, tangential reenforcing scleral bites; 4, point of tie on the anterior surface of the conjunctiva; 5, tendon stump; 6, edges of conjunctival wound. (From Peter, "Extraocular Muscles," 2nd Ed., Lea & Febiger, 1936.)

at (3) after the sutures have been passed through the capsule and conjunctiva. In all other instances it is sufficient to make the tie after the scleral bite at (2). In either case the tie is made on the conjunctival surface. Both eyes are bandaged for five days, although dressings are changed daily after the first dressing at the end of forty-eight hours. No attempt should be made to remove the suture before the seventh day, at which time it can readily be removed after several instillations of cocaine.

PARALYTIC SQUINT

Tendon Transplantation of the Vertical Muscles. In complete paralysis of the superior and inferior recti, as a rule, two surgical procedures are indicated. If the corresponding or yoke oblique in the opposite eye overacts, the first surgical step is tenotomy of the oblique. For example, in paralysis of the right superior rectus, the left inferior oblique is apt to be overactive, especially if the paralysis is of long standing. Tenotomy of the oblique, as a rule, will not restore the two visual axes to the same horizontal level. Nevertheless, it is the first step to practice and eventually will lead to complete recovery. By this operation, the yoke muscles are placed on a par, in that the three functions of each are weakened. It cannot be said that the elevating power of an oblique is equal to that of its co-worker, the superior rectus. In complete paralysis of the superior rectus, therefore, there will be a residual hypotropia of about 10 degrees. This calls for a shortening operation, and, in most instances, an advancement of the superior rectus tendon is indicated. By weakening the left inferior oblique, however, either by complete tenotomy or by a lengthening of its tendon, both eyes are placed in approximately similar positions, in weakness of left rotation, in tortional action to the left and in defective elevating power. To correct the residual right hypotropia, advancement of the right superior rectus is the operation of choice.

The technique of advancement of the right superior rectus differs somewhat as to whether it is the second procedure to correct a complete paralysis of the muscle or the primary operation to overcome a partial paralysis or paresis. In the former instance, as tortional weakness and rotation to the left have been equalized by tenotomy of the oblique, elevation of the right eye alone should be considered, and an advancement of the right superior rectus should be in line with the original insertion and should not be displaced either to the right or to the left.

The author's advancement technique, as described and outlined in Figure 3, is ideal. Two scleral bites should be used. (Fig. 3, points 2 and 3.) This will insure against slipping of the sutures during the healing process. Those who are partial to the O'Connor cinch may elect to practice this in lieu of an advancement. In the author's experience a cinch operation is not the operation of choice in a paralyzed muscle. For the same reason, tucking also is contradicted unless the hypotropia is of low degree.

If *paresis* of the superior rectus is moderate and the muscle has some contractile power, the yoke inferior oblique may not require any attention, and an advancement of the superior rectus may be sufficient to restore the horizontal balance. In this instance, the operator should pay due attention to the tortional weakness to the left or right. A well placed advancement may correct the vertical defect or hypotropia and at the same time may overcome the tendency to extortion. To correct both phases of superior rectus weakness, the advanced tendon should be reattached near the limbus and to the *temporal* side of the former insertion. How far to the side the advancement should be placed can only be determined by the degree of extortion and by experienced judgment.

In complete or in partial paralysis of the superior rectus the accompanying and residual exophoria or exotropia which may

be present will call for readjustment of the internal or external rectus for its correction.

Tendon Transplantation in Paralysis of an Inferior Rectus. Diplopia in the lower field is more annoying than in the upper or lateral fields. Paralysis of an inferior rectus consequently calls for accurate surgery. If the yoke superior oblique is overactive, a complete tenotomy may be indicated. This is executed through a skin incision beneath the brow and over the trochlea where the superior oblique tendon can be reached.

If the oblique is not manifestly in spasm, the first surgical procedure should be an advancement of the paralyzed inferior rectus. Reattachment should be close to the limbus and to the outer side of the original insertion. Such advancement will overcome considerable hypertropia and will tend to overcome excessive intortion. Any residual hypertropia may be corrected by a recession of the superior rectus on the same side.

Paralysis of the Superior Oblique. Shortening of the superior oblique can be done but is attended by some difficulty. The superior rectus should be detached in order to reach the tendon of the superior oblique. The intimate relation between the superior rectus and superior oblique where these muscles cross, the close relation between the vorticose veins and the tendon of the oblique, and the care with which the superior rectus must be reattached, render the operation difficult and dubious as to end results. Because of the complications and the extensive postoperative adhesions, in the author's judgment, the procedure is a questionable one. Those who are interested will find the technique described by Wheeler.[3]

The surgeon's attention, therefore, should be directed to the inferior rectus of the opposite eye. Tenotomy of a vertical rectus can only tend to complications and should never be practiced. In lieu of tenotomy, the tendon of the inferior rectus should be receded by careful technique. Even a recession of a vertical rectus cannot be done with the same freedom as that of the internal or external rectus. A recession of 2 or 3 mm., as a rule, is adequate to correct the hypertropia of the contralateral eye caused by superior oblique paralysis. To accomplish this the author's recession suture is most applicable. A recession of a few millimeters will tend to weaken extorsion, but this can further be influenced by slight displacement toward the nasal side of the original insertion. The cyclophoric disturbance in the contralateral eye, therefore, will approximate that of the paralytic side and a tendency to esophoria, which usually accompanies oblique paralysis is slightly improved. Any appreciable residual esophoria which may require attention, can be overcome by shortening of an external rectus or recession of an internal rectus tendon.

Inferior Oblique Paralysis. The tendon of the inferior oblique can be reached through a skin incision along the inferior orbital rim over the origin of the muscle on the floor of the orbit. The same surgical procedures which may be indicated in paralysis of its yoke muscle, the superior rectus, are applicable and may be followed. For example, if the contralateral superior rectus shows spasm or overaction, a recession of the superior rectus tendon is indicated. *As stated previously, complete tenotomy of a superior or inferior rectus cannot be considered.* A restraining suture is always indicated. A proper recession will place the yoke superior rectus in approximately the same disadvantageous position as the paralytic inferior oblique and vertical, lateral and torsional diplopia will be reduced appreciably. Residual hypotropia, if any, is corrected by shortening the tendon of the inferior oblique. Instead of tenotomizing the tendon, after it is exposed through a skin incision along the inner orbital rim and an opening into the *fascia orbitale*, a "00" ten day chromicized catgut suture is carried through the tendon, after which it is freed from its origin, brought out through the fascial opening and attached to the periosteum of the

orbital rim. The degree of shortening is one of surgical judgment and experience.

End Results. The reader may ask, what are the average end results of tendon transplantation in paralysis of the superior and inferior recti and the two obliques?

The average results are good if the operative procedures recommended are followed by correct refraction, moderate prismatic correction for residual hyperphoria and finally by orthoptic training. Most patients can be made reasonably comfortable. Much will depend upon the duration of the palsy, accuracy of technique and to some extent upon the temperament of the individual, i.e., the desire to cooperate with the surgeon.

Paralysis of External and Internal Recti. In discussing "*tendon transplantation*" much interest centers in the rather extensive transplantation required in lateral palsies. Each eye has two upward and two downward rotators. Laterally, there is but a single muscle to the right and to the left, aided moderately in each direction by two synergists. In concomitant squint and in paralytic squint of a vertically acting muscle, tendon transplantation merely increases or diminishes the normal physiologic action of the muscle. In lateral palsies an attempt is made to so change the insertions of certain muscles, as to alter their normal actions and create new ones. To be more specific, transplantation of part of the superior and inferior recti tendons, is an effort to transfer vicariously the function of external rotation from the external rectus to the superior and inferior recti. True enough, external rotation is not a wholly new function for these muscles, but it is a function which can be exercised only in secondary positions of the eyes. To accomplish external rotation from "eyes front" or in primary position, by changing and broadening the attachments of the two vertical recti, it is first necessary to place the eye on the paralyzed side in a position to obtain a position of "eyes front." Following this, when properly transplanted, the vertical recti reënforce the obliques in the action of external rotation when this is required.

Complete paralysis of an external rectus will rarely admit of perfect centering in primary position. This can only be accomplished by a free resection and advancement of the paralyzed muscle and recession of the internal rectus on the same side. Worth's advancement technique is the operation of choice from the external rectus. The author's or any other satisfactory recession technique may be employed for the internal rectus. These two procedures may insure a fair degree of single binocular vision in primary position and in convergence. It will not admit of single binocular vision in the field of action of the paralyzed muscle. The technique, therefore, is inadequate in most instances to yield satisfactory cosmetic results and will fall far short of perfect functional activity.

The O'Connor Transplantation or Its Modifications. To obtain external rotation beyond "primary position," the tendons of the superior and inferior recti are split in order to leave a strand sufficiently adequate to continue the vertical action of these muscles and to broaden the outward attachment of the tendon so as to create outward rotation. For this purpose the writer has modified the O'Connor technique to meet the requirements.

General anesthesia is necessary (preferably avertin), to give the operator freedom of action without restraint and to reduce the operative trauma and postoperative reaction to a minimum.

The conjunctival and capsule incision is carried from the superior to the inferior rectus about 1 cm. from the limbus. A Worth advancement of the external rectus is completed up to the point of tying the sutures, which is left to the last part of the operation. Recession of the internal rectus may be the second step or this may be the last of the muscles to be operated on. There are good reasons for this choice, chief of which are the danger of disturbing the delicate scleral bites if it is practiced as the

second step, and the danger of disturbing the delicate transplants if it is done last. In either instance manipulations should be careful and precise.

technique is followed. In the writer's experience, varying amounts of rotation have been obtained, from 10 to 45 degrees. In each instance of indifferent external rota-

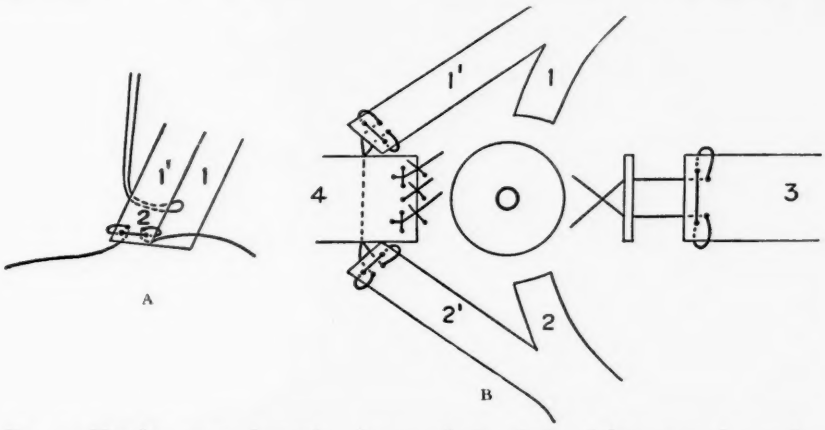


Fig. 4. Tendon transplantation for paralysis of the right external muscle. A, preparation of superior rectus transplant. 1, inner half of superior rectus; 1′, outer half of superior rectus elevated on muscle hook; 2, whip stitch placed in the outer half to be transplanted. B, diagrammatic sketch of tendon transplantation of outer halves of superior and inferior recti; recession of internal rectus and advancement of external rectus. 1, superior rectus; 1′, superior rectus transplant; 2, inferior rectus; 2′, inferior rectus transplant; 3, receded internal rectus; 4, advanced external rectus. (From Peter, "Extraocular Muscles," 2nd Ed., Lea & Febiger, 1936.)

The transplants are carefully dissected and should include one-half of each tendon or a trifle more. The tendons should be exposed and the splitting should continue back at least 10 to 11 mm. above and as far back in the inferior tendon as the capsule will permit. Sutures (No. 1 white silk, waxed) are introduced as shown in the diagram. (Fig. 4.) By rotating the eyeball to the temporal side, they can be attached to the stump of the external rectus without too much stretching. After both transplants are in place, the Worth advancement is completed by tying the three sutures. The conjunctiva and capsule above and below the external rectus and over the internal rectus are closed with running sutures.

Both eyes are closed with a compress bandage for five days, and redressed on the seventh day, after which the unoperated eye is allowed to be open.

What are the end results? What degree of external rotation can be obtained? Can one obtain single binocular vision?

The answers to these questions must be predicated on the care with which the tion, some flaw in the technique can be recalled. The procedures are difficult and require precision and care. Binocular vision is possible in most instances. There will be a certain percentage of failures in the matter of external rotation. On the whole, it is the only operation of real value to bring about a maximum degree of cosmetic and functional cures, and the operation is sufficiently encouraging to warrant it's performance in all cases of external rectus paralysis. Experience, however, is a most important factor. This procedure should not be attempted by one whose training and experience are inadequate.

For internal rectus paralysis, the technique is similar. The end results are good cosmetically, but it is difficult to restore full converging power.

Paralysis of the third nerve offers an opportunity, under certain conditions, of converting a depressor muscle into an internal rotator. If the eye is amblyopic or if the paralysis occurs in a young patient (not over 15 years of age) so that suppression can be acquired, the eye can be

centered in the horizontal meridian, internal and external rotations can be established, and the ptosis can be corrected by suitable tendon transplantation. The first step in the series of operations necessary to bring about really good cosmetic results is transplantation of the tendon of the superior oblique, as it escapes from the trochlea, to the point of attachment of the internal rectus. The external rectus is receded to bring about careful centering of the eye. Both operations are performed at the same session. After the reaction has subsided, a final step in the surgical procedures is the correction of the ptosis either by shortening of the levator of the lid by the Blaskovicz operation,* if this tendon is not completely paralyzed or by a Hunt-Tansley operation. For minute details of the technique, the reader is referred to a description of the author's technique found in "Extra-Ocular Muscles" (second Edition, page 328) or in the original report by the author.[4]

The results are so satisfactory that the procedure is recommended to those who are experienced in ocular muscle surgery.

SUMMARY

1. The author has included, under tendon transplantation: (a) the simple forms of recessions and advancements which, as a rule, are practiced in concomitant squint; (b) the more complicated tendon recessions and advancements which are required in paralytic squint; and (c) transplantations which are indicated in special instances, in which a transplanted tendon creates an entirely new function for one or more of the ocular muscles.

2. In any form of tendon transplantation (a) refinement of technique, (b) the avoidance of unnecessary tissue trauma, and (c) precision in every detail, are factors which will insure good results, a minimum amount of cicatricial tissue formation, and an excellent cosmetic appearance of the local wound area.

3. In convergent concomitant squint, the choice of operation should be based on the degree of deviation of the visual axes, and on the dominance or overaction of internal rotators or underaction of external rotators. In divergent squint the choice is determined in a similar way.

4. Paralytic squint calls for careful analysis of the diplopia field, the limitation of movement and the special needs of each form of ocular muscle palsy. The end results, in paralysis of the vertical recti and obliques, justify the methods set forth in the text.

5. Correction of external rectus paralysis, by the O'Connor or similar technique, is the only type of operation which will restore wholly, or in part, external rotation. In moderate paresis of this muscle, a full advancement of the external rectus and a moderate recession of the internal rectus may be adequate to give the patient a reasonable degree of binocular functional activity.

6. The creation of a new muscle function by radical tendon transplantation, as for example, changing the action of depression of a superior oblique to that of internal rotation of the eyeball, is possible. The faculty of binocular rotation to right and left, in the horizontal plane, will depend only on the careful adjustment of the superior oblique as an internal rotator, and corresponding readjustment of the external rectus.

* The Blaskovicz operation consists in shortening of the levator of the lid, the procedure being carried out from the conjunctival surface, thereby avoiding any defect of the skin surface by scar tissue.

REFERENCES

1. Jameson, P. Chalmers. The surgical entity of muscle recession. *Arch. Ophth.*, 6: 329, 1931.
2. Peter, Luther C. The Extra-Ocular Muscles. Phila., 1936. Lea & Febiger, Second Edition.
3. Wheeler, John. *Tr. Am. Ophth. Soc.*, 32: 237, 1934.
4. Peter, Luther C. *Tr. Am. Ophth. Soc.*, 31: 232, 1933.

CERTAIN POSTOPERATIVE COMPLICATIONS OF CATARACT OPERATIONS*†

WITH ESPECIAL REFERENCE TO A STUDY OF 1,004 OPERATIONS

CONRAD BERENS, M.D. AND DONALD W. BOGART, M.D.

Chairman, American Board of Ophthalmology

Assistant in Ophthalmology, New York University School of Medicine

NEW YORK CITY

COMPLICATIONS following cataract operations are serious principally because they may result in complete or partial loss of visual acuity. Because many of these complications may be prevented and a comparison of the results of intracapsular and extracapsular extraction in the hands of the average surgeon seemed desirable, we have analyzed the records of cataract extractions performed at the New York Eye and Ear Infirmary during the past two years.

TABLE 1

COMPARISON OF POSTOPERATIVE COMPLICATIONS FOLLOWING INTRACAPSULAR AND EXTRACAPSULAR CATARACT EXTRACTION IN 1,004 PATIENTS

Complications	Intracapsular (302 Patients)	Extracapsular (702 Patients)
Postoperative eversion of the cornea	1	1
Hemorrhage into the anterior chamber.	12 (4 per cent)	10 (1.4 per cent)
Expulsive hemorrhage	2 (0.7 per cent)	3 (0.43 per cent)
Bullous keratitis	1	1
Keratitis	1	2
Ulcer of cornea	2	4
Iritis	2	0
Iridocyclitis	0	7
Secondary glaucoma	2	7
Prolapse of iris and ciliary body	1	0
Prolapse of iris	2	6
Dense secondary membrane	5	38
Loss of vitreous	30 (9.9 per cent)	63 (9.0 per cent)
Prolapse of vitreous	1	2
Endophthalmitis (large vitreous opacities)	5	3
Detachment of the retina	1	3
Suppurative intraocular infection	0	1
Pneumonia	0	1
Detachment of the choroid	1	0
Hernia of vitreous	1	0
Uveitis	1	0
Choroiditis	0	2
Capsular remains in anterior chamber	0	1
Mental disturbance	1	1
High astigmatism (over 6 D)	1	1
Totals	72 (24 per cent)	157 (22.3 per cent)

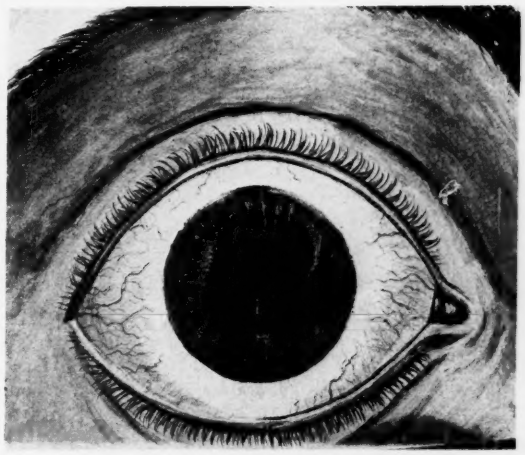


FIG. 1. Striate keratitis with blood staining in the cornea.

A total of 1,004 clinic patients (Table 1) were operated upon for cataract by surgeons, assistant surgeons and house surgeons. Of this number there were 702 extracapsular extractions (70 per cent) performed and 302 intracapsular extractions (30 per cent). Of the extracapsular extractions 119 were attempted intracapsular extractions. Mature senile, immature, hypermature, complicated, perinuclear, congenital, traumatic, posterior cortical, anterior polar, posterior capsular and coerulean cataracts were included in this group. In evaluating the results, it must be remembered that the extracapsular as well as the intracapsular extractions were often performed by inexperienced surgeons and

* Presented before the Section on Ophthalmology, American Medical Association, June 13–17, 1938.

† From the Department of Research, New York Eye and Ear Infirmary. Aided by a grant from the Ophthalmological Foundation, Inc.

that frequently preoperative complications were present.

The average age of the patients in the extracapsular group was 62, although the ages ranged from 22 years to 85 years. Among the patients on whom intracapsular extractions were performed the average age was 65, the ages ranging from 27 to 90. The loop was used to extract four (1.4 per cent) of the lenses in the intracapsular operations and 34 (5 per cent) in the extracapsular operations.

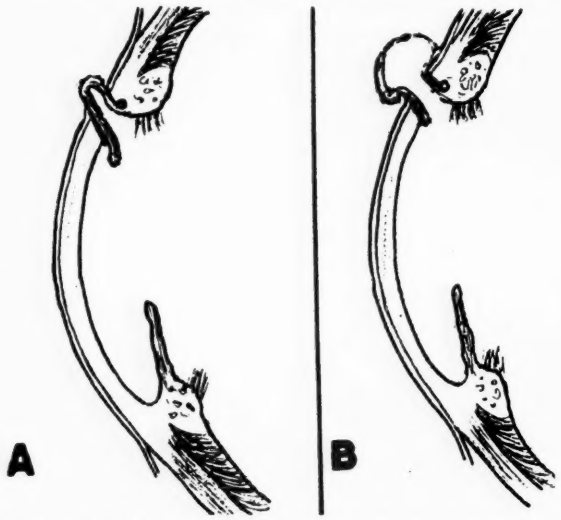


FIG. 2. A, prolapse of the iris in the form of a hernia. B, large hernia of the iris which has produced an extensive cystoid cicatrix.

Among the complications frequently occurring after cataract extraction are:

I. COMPLICATIONS INVOLVING THE CORNEA

STRIATE OPACITY OF THE CORNEA

Cause. It is believed that striate keratitis (Fig. 1) may be caused by (1) folding of the deeper layers of the cornea, especially Descemet's membrane; (2) difference in tension in the vertical and horizontal diameters of the cornea, resulting from incision near the corneal margin;[1] (3) injury to Descemet's membrane during delivery of the lens, especially when a large hard lens has been forced through a small incision with resulting aqueous infiltration of the corneal substance; and (4) trauma of the endothelial cells covering Descemet's membrane.[2] Elschnig[3] found that it rarely follows forceps extraction although it is more common following capsulotomy. These opaque lines are not accompanied by marked inflammatory reaction and usually disappear within a few days.

Incidence. Striate keratitis was seen by Appleman[4] in five of 100 patients on whom intracapsular extraction was performed. Slocum[5] reports that striate keratitis occurred in seven of the 882 operations for senile cataract.

Prevention. A large section, care in employing instruments and the use of half normal physiologic saline solution, warmed to body temperature, apparently are aids in preventing the development of the traumatic striate opacity.

Treatment. Hot applications and the instillation of dionin seem to be beneficial.

II. COMPLICATIONS INVOLVING THE IRIS

INCARCERATION AND PROLAPSE OF THE IRIS

Prolapse of the iris, which usually occurs within three or four days after operation, may be heralded by the fact that the patient complains of slight pain. Iris prolapse may appear in the form of a slight hernia (Fig. 2A) or a large cystoid cicatrix (Fig. 2B) may develop.

Cause. Prolapse of the iris in unsutured wounds may be caused by (a) trauma, (b) defective incisions, and (c) omission of iridectomy.[6] Pallares Lluesma[7] attributed a hernia of the iris to the traction of a pterygium.

Incidence. Prolapse of the iris alone was observed in six of our patients on whom extracapsular extraction was performed and in two of the intracapsular cases. The ciliary body and the iris were prolapsed in one of our intracapsular cases. In one of these patients the herniated iris was noted five days after the operation, but the eyes had been irritated for several weeks postoperatively; final vision was $^{20}/_{40}$. The incidence of prolapse of the iris in this series of cases and in the practice of other ophthalmologists is tabulated in Table II.

Prevention. If the incarceration is slight, usually no serious difficulty results, especially if the iris is covered by conjunctiva. If the sutures are holding, it may be safe to apply eserine ointment and allow a few days to elapse before operation. Cystoid scars and small beads of prolapsed iris have been reduced by applying the actual cautery[8] or electrocoagulation,[9] but the wound always should be covered with a conjunctival flap. However, the actual cautery is not recommended by us for the treatment of any form of incarcerated or prolapsed iris. The application of trichloracetic acid as suggested by Bettman and Barkan[19] is valuable for the treatment of certain types of iris prolapse.

If prolapse is *extensive* and not covered by conjunctiva, a small iridectomy should be performed and the wound closed with sutures.

If prolapse is *extreme* a conjunctival flap should be made by dissecting from the cornea upward; after excising the protruding iris the flap is drawn downward over the cornea and sutured below. However, if sutures are in place, the wound should be gently opened, the iris grasped with iris forceps and iridectomy performed.

TABLE II
INCIDENCE OF PROLAPSE OF THE IRIS AFTER CATARACT EXTRACTION

Surgeon	No. of Cases	Iris Prolapse		Operation
		No.	Per Cent	
Bothman[10]	1000	17	1.7	Indian
Dunphy[11]	1821	9	0.4	Combined
	298	4	1.3	Simple
	441	0	....	Intracapsular
Parker[12]	100	1	1	Combined
	100	6	6	Simple
	100	2	2	Knapp-Török
Elschnig's Clinic[13]	1000	22	2.2	Capsulotomy
	44	4	10	Smith
	35	3	9.5	Stanculeanu
	46	1	1.5	Török
	76	2	2.7	Barraquer
Shipman[14]	100	2	2	Shipman
Beach and McAdams[15]	100	4	4	Verhoeff[16]
Parker[17]	150	2	1.3	Combined
	150	6	4	Simple
	150	4	2	Knapp-Török
Gailey[18]	200	6	3	4 Extracapsular 2 Intracapsular
Berens and Bogart	302	3	1.0	Intracapsular
	702	6	0.9	Extracapsular
Totals	6915	104	1.5	

IMPLANTATION CYSTS

Implantation cysts in the iris occurring after cataract extraction have been reported by Greeves[20] and one has been reported in the posterior chamber by Krug.[21]

III. COMPLICATIONS INVOLVING THE ANTERIOR CHAMBER

A. HEMORRHAGE INTO THE ANTERIOR CHAMBER (HYPHEMIA)

Postoperative hemorrhages may appear in the anterior chamber with or without pain; they usually originate from the sclera or from the iris, rarely from the conjunctiva.

Incidence. Hyphemia developed in twelve (4 per cent) of the intracapsular extractions and in ten (1.4 per cent) of the extracapsular extractions. Vail[22] found

TABLE III
INCIDENCE OF HYPHEMIA FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Hyphemia		Operation
		No.	Per Cent	
Bothman[10]	1000	36	3.6	Indian
Parker[12]	300	1	0.33	Combined
Shipman[14]	100	6	6	Shipman
Beach and McAdams[15]	100	5	5	Verhoeff[16]
Vail (D. T. Sr.) 22*	890	11	1.23	
		2	0.4	Intracapsular (1 Smith)
		9	2.1	Extracapsular
Vail (D. T. Jr.)[22*]	510	27	5.29	
		21	6.9	Intracapsular (4 Smith)
		6	2.8	Extracapsular
Appleman[4]	100	4	4	Knapp-intracapsular
Giri[23]	16			Combined extracapsular with capsulectomy
	245	4	1	Combined intracapsular
	123			Simple intracapsular
Gailey[18]	200	9	4.5	Extracapsular
		4	2	Intracapsular
Berens and Bogart	302	12	4	Intracapsular
	702	10	1.4	Extracapsular
Totals	4588	129	2.8	

* D. T. Vail, Sr. employed corneolimbal incision; D. T. Vail, Jr., conjunctival flap or suture.

that hyphemia occurred in 7.59 per cent of all cases of cataract extraction. (Table III.)

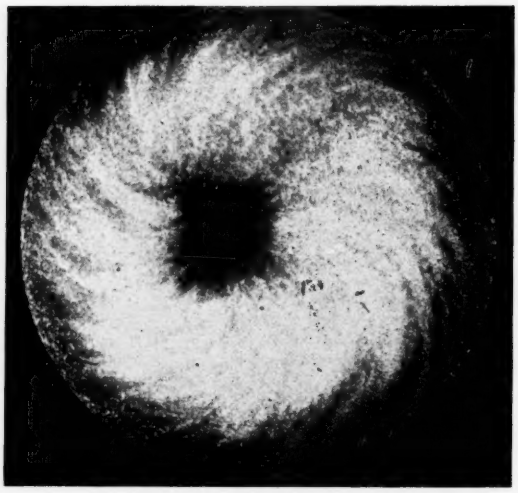


Fig. 3. Detachment of the choroid in a girl eighteen years of age.

Prevention. To prevent the occurrence of hemorrhages careful preliminary studies are necessary in order to eliminate foci of infection or to raise resistance to them. Coagulation and bleeding time should be normal. Diabetes and high blood pressure should be controlled and syphilis excluded. The patient should not be permitted to cough or strain. At the time of dressing, the eyes, while closed, should be gradually light adapted; the healthy eye should be opened first. After the eyelashes of the eye, which has been operated upon, are free from secretion, the patient should be instructed to open both eyes and the lower eyelids should be drawn down gently. One must avoid attempting to observe the upper border of the wound or touching the upper eyelid. The use of strong miotics should be avoided after the primary instillation, unless prolapse of the iris tends to develop, because spreading the iris tissues and dislodging clots may cause hemorrhages.

Treatment. Large hemorrhages may protrude between the lips of the wound. It may be necessary to remove part of the clot in order to permit the wound to close. If repeated hemorrhages occur, coagulation, bleeding time and the number of platelets should be studied. Calcium gluconate injected intravenously, normal horse serum, snake venom and brain extract[24] may be administered. It is claimed by Thilliez[25] that the subcutaneous injection of epinephrine hydrochloride and ergot will arrest hemorrhage promptly. Retrobulbar injections of epinephrine hydrochloride may also be used. Opinions differ as to whether or not procaine hydrochloride and epinephrine hydrochloride, injected retrobulbarly, have a tendency to increase the incidence of intraocular bleeding. If increased tension develops, it may be necessary to wash out the anterior chamber and remove part of the clot with forceps.

When blood must be removed from the anterior chamber, injection of sterile air is advocated by Abramowics.[26] He employs a lacrimal syringe, which is held over an alcohol flame while being filled with air. The air is then introduced into the anterior chamber.

B. DEEP ANTERIOR CHAMBER

A deep anterior chamber immediately after cataract extraction may be caused by fluid vitreous. Elschnig[3] believes that inflating the anterior chamber with sterile air will correct this condition.

IV. DETACHMENT OF THE CHOROID

Subchoroidal exudates, which occur quite frequently, may be seen as dark, grayish, round masses, usually extending into the vitreous from the anterior part of the choroid. These exudates often absorb with surprising rapidity. In our practice, these cases have not been accompanied by serious visual disturbances. Although the choroid seemed to be completely detached (Fig. 3) in a girl 18 years of age, after ten days no ophthalmoscopic evidence of detachment was observable.

Cause. The cause of subchoroidal exudates is not necessarily high blood pressure, but lowering of intraocular pressure may be an important factor.

Incidence. Detachment of the choroid was observed in only one patient on whom intracapsular extraction had been performed. However, in this series of 1,004 extractions routine ophthalmoscopic examinations were not made for several weeks postoperatively. Detachment of the choroid occurs at the time of operation in almost all cases according to O'Brien.[27] Hill[28] found detachment of the choroid in 75 per cent of cases within twenty-four hours after intracapsular extraction. He does not feel justified in using the ophthalmoscope immediately after operation; by examining twenty-four hours later, he believes that he arrives at a percentage considerably lower than actually obtains. The incidence of detachment of the choroid is much lower according to Fuchs (4.7 per cent)[29] and Barkan (4.5 per cent).[30]

Prevention. Retrobulbar injections of procaine and adrenalin have been advocated to prevent this complication but the efficacy of this treatment is questioned by Jensen.[31] Manes,[32] who believes that detachment of the choroid is a favorable complication, attempts to provoke it by gentle pressure on the inferior segment of the eyeball with a strabismus hook.

Treatment. The following treatment is recommended: (1) rest in bed with the head elevated; (2) measures to improve the coagulation of the blood; and (3) sedatives.

V. EXPULSIVE INTRAOCULAR HEMORRHAGES

Fortunately, expulsive intraocular hemorrhage is a rare complication. It is recognized by the sudden and profuse flow of blood, which forces out the contents of the eyeball, replacing them with clotted blood. The choroid and retina are torn from the sclera.

Cause. Postoperative histologic examination seems to indicate that the hemorrhage is caused by ruptured choroidal veins. In the six expulsive hemorrhages observed by Gailey,[18] the blood pressure was low for the patient's age, all well below 185. Gailey questions whether intraocular tension is not as great a factor in hemorrhage as arterial hypertension. In a private patient recently operated upon for cataract by one of us (C.B.), the blood pressure was 160/80. Expulsive hemorrhage occurred the day after operation. Vision following the Verhoeff scleral puncture was faulty light projection.

Incidence. Clapp[33] gives the incidence as one case in 1,000. Expulsive hemorrhage occurred in two (0.7 per cent) of our intracapsular extractions and in three (0.43 per cent) of our extracapsular extractions. (Table IV.) The eye was eviscerated in the three patients in the extracapsular group while in the intracapsular group the eye was enucleated in one patient and in the other, the eye was eviscerated.

TABLE IV
INCIDENCE OF EXPULSIVE HEMORRHAGE AFTER CATARACT EXTRACTION

Surgeon	No. of Cases	Expulsive Hemorrhage		Operation
		No.	Per Cent	
Bothman[10]	1000	10	1.0	Indian
Dunphy[11]	1821	4	0.21	Combined
	298	1	0.33	Simple
	441	0	0	Intracapsular
Parker[36]	1421	3	0.21	
Barraquer[37]	1000	1	0.1	Phacoerisis
Nugent[2]	2200	17	0.78	
Wolfe[38]	100	1	1	Barraquer
Gailey[18]	200	1	0.5	Extracapsular
Berens and Bogart	302	2	0.7	Intracapsular
	702	3	0.43	Extracapsular
Total	9485	43	0.45	

Prevention. It is reported that in many clinics, for example, Elschnig's at Prague, from 300 to 500 c.c. of blood is withdrawn from every patient with high blood pressure in order to prevent expulsive hemorrhage.[34] Gifford,[34] however, questions the efficacy of this treatment. Careful preoperative examination is important. In patients, who have lost one eye from expulsive hemorrhage, a trephine operation may be performed to accustom the eye to lowered tension;[35] our preference is for iridocorneosclerectomy.[50] Nugent[2] reported that the percentage of choroidal hemor-

rhages was reduced from 2 to 0.9 per cent by lowering high intraocular tension or by performing an extracapsular operation.

Treatment. Following expulsive intraocular hemorrhages, the eye is usually destroyed. A light compressing dressing and sedatives may be administered, but treatment, except that advised by Verhoeff,[39] does not seem to prevent complete loss of vision. Woodruff[40] advises not to remove the eye immediately following expulsive hemorrhage, for at times a normal eyeball may be retained. It is stated that postoperative intraocular hemorrhage may be arrested by the subcutaneous injection of epinephrine hydrochloride and ergot.[25] In addition to the injection of ergot and epinephrine hydrochloride (10 minims), injection of a solution of posterior pituitary (10 minims) two or three times a day, is recommended by Ziegler[41] as a prophylactic measure. This may also be used to control bleeding. Posterior sclerotomy over the site of the hemorrhage saved one eye operated upon by Verhoeff[39] and the eyes of two patients operated upon by Vail;[42] one of these patients had final visual acuity of $^{20}/_{25}$ and the other $^{20}/_{200}$. This treatment advised by Verhoeff seems to be the only practical method of saving useful vision in these eyes. Unfortunately, in the cases reported in our series this operation was not performed and all five eyes were lost. This operation has been performed by one of us (C. B.) recently and the patient now has fair light projection.

VI. DETACHMENT OF THE RETINA

According to Woodruff[40] retinal detachment following cataract extraction is practically incurable. He obtained cures in only 18 per cent of the cases subjected to operation.

Cause. Retinal detachment is not necessarily a complication of the operation but may be caused by anterior choroiditis of tuberculous or focal infectious origin. Myopia and loss of vitreous were the important factors in thirty cases of retinal detachment reported by Shapland.[43]

Incidence. Of our series of 1,004 patients, retinal detachment occurred in three patients on whom extracapsular extractions were performed and in one patient of the intracapsular series. In the extracapsular cases, vision in one case was $^{4}/_{200}$; in another, hand movements; and in the third patient, vision was not recorded. In the intracapsular case final vision was $^{20}/_{200}$. In Table v our results and those reported by other surgeons are tabulated.

TABLE V
INCIDENCE OF DETACHMENT OF THE RETINA AFTER CATARACT EXTRACTION

Surgeon	No. of Cases	Detachment of the Retina		Operation
		No.	Per Cent	
Beach and McAdams[15]	100	2	2	Verhoeff[16]
Knapp[46]	500	6	1.2	Intracapsular
Appleman[4]	100	3	3	Intracapsular (Knapp)
Peter[47]	112	2	2	Intracapsular
Slocum[5]	882	9	1.03	Conjunctival bridge and suture
Berens and Bogart	302	1	0.34	Intracapsular
	702	3	0.43	Extracapsular
Total	2698	26	1	

Treatment. Cruise[44] reported successful results following the Safar operation. A cure by the same method is also reported by Hine[45] in a case in which detachment occurred after a needling operation. One of our most successful results was obtained in a case of retinal detachment following cataract extraction. The Gonin method, combined with aspiration of the subretinal fluid, resulted in closure of a large hole, complete reattachment and final visual acuity of $^{20}/_{30}$. Detachment also occurred in a diabetic patient after cataract extraction and a fair visual result was obtained by operation.

VII. GLAUCOMA AFTER CATARACT EXTRACTION

Hypertension immediately following cataract operation is observed infrequently, even though glaucoma may have been present before operation as it was in nine

(3 per cent) of the intracapsular cases and twenty-seven (3.9 per cent) of the extracapsular cases.

When the lens swells rapidly, as for example in cataract caused by dinitrophenol or dinitrocresol, removal of the lens apparently cures the glaucomatous condition. Tension is likely to be increased if the cataract is immature and soft lens matter remains, especially in congenital and juvenile cataracts in which complete discission of the lens[48] has been attempted and only the anterior capsule has been incised.

Incidence. The incidence of postoperative glaucoma in our cases is lower than we believe it would be if preoperative and postoperative tension had been measured routinely.[49] Of the 1,004 extractions performed at the New York Eye and Ear Infirmary, secondary glaucoma occurred in two of the intracapsular extractions and in seven of the extracapsular extractions. The incidence of secondary glaucoma following cataract extraction as reported by certain surgeons is tabulated in Table VI.

TABLE VI
INCIDENCE OF SECONDARY GLAUCOMA FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Secondary Glaucoma		Operation
		No.	Per Cent	
Parker[12]	300	2	0.66	Knapp-Török
Shipman[14]	100	6	6	Shipman
Fox[51]	856	14	1.5	Extracapsular
		10		Discission
Knapp[46]	500	14	0.28	Intracapsular
Slocum[5]	882	4	0.5	Conjunctival bridge and suture
Berens and Bogart	302	2	0.67	Intracapsular
	702	7	1.0	Extracapsular
Total	3642	59	1.62	

Prevention. Some cases of secondary glaucoma following cataract extraction possibly may be prevented by thorough preliminary study of the patient, including slit lamp examination for possible iridocyclitis, and by complete removal of soft lens matter and careful toilet of the wound.

Treatment. In glaucoma caused by swelling of the lens or by lens matter, removal of the lens or lens particles improves the condition. If chronic simple glaucoma, which is not caused by swelling of the lens, is present, a filtering cicatrix may develop. This tendency should be favored by massage, if there is a satisfactory conjunctival flap, and by excision (with a punch[50]) of some of the scleral lip of the wound which should be made with a keratome at the time of operation. Hypertension caused by iridocyclitis, may respond to the use of mydriatics and paracentesis or aspiration[52] of the anterior chamber. (Fig. 4.) Hypertension, produced by chronic iridocyclitis, and a wound in which the vitreous, lens capsule and iris are incarcerated, may be extremely rebellious to treatment.

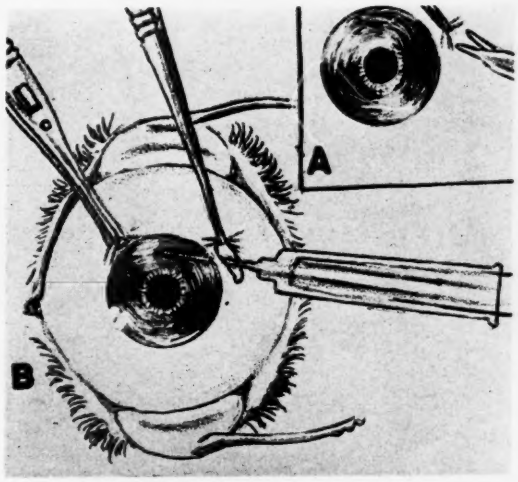


FIG. 4. Aspiration of the anterior chamber. A, incision in the conjunctiva to which iodine is applied. B, subconjunctival introduction of the needle with slight rotary motion, keeping the opening in the needle up. The surgeon steadies the syringe while the assistant slowly withdraws the plunger.

If hypertension persists in spite of the use of miotics, mydriatics and aspiration or paracentesis and no cause in the nature of a focal infection or other disease may be found, operation must be performed. If a simple extraction has been performed, we usually prefer iridocorneosclerectomy.[50] If there is a coloboma of the iris, we perform corneosclerectomy over one pillar combined with iridencleisis. (Fig. 5.) If this

does not suffice, the operation is performed on the other pillar. The wound may be reopened under a conjunctival flap with a

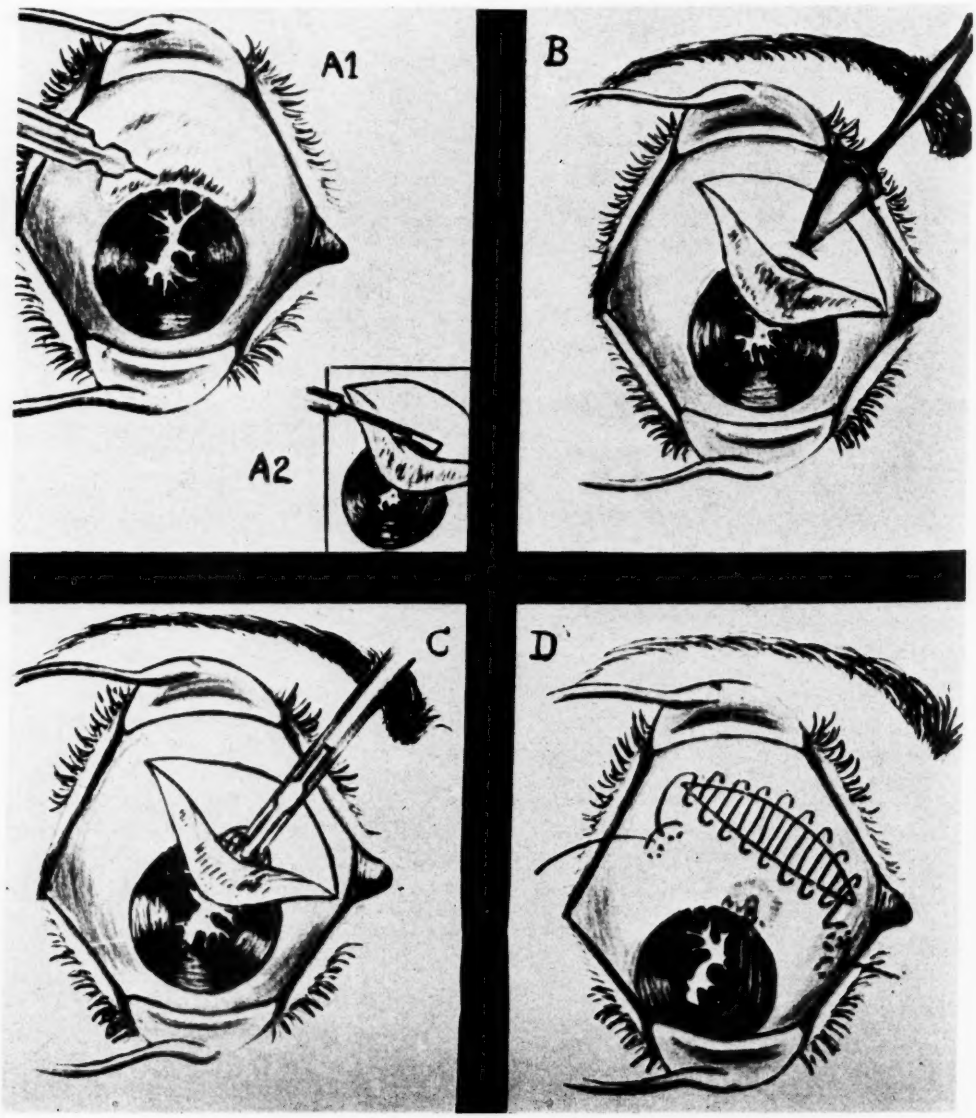


FIG. 5. Iridencleisis combined with corneosclerectomy. A, 1, conjunctiva and Tenon's capsule ballooned over nasal pillar with procaine-adrenalin solution; 2, conjunctival and Tenon's capsule incised 6 mm. above cornea and turned down over the cornea. The corneal layers being separated by sharp dissection for a distance of 1.5 mm. B, incision with a keratome, hollow ground on the under surface, entering the sclera 1.5 mm. from the limbus over the nasal pillar. C, special punch used to excise the anterior lip of the scleral wound and 0.5 mm. of the cornea, after making an incision toward the cornea, at both ends of the scleral wound with Stevens scissors. D, iris pillar drawn out under the conjunctiva and wound in conjunctiva and Tenon's capsule being closed with a running suture of oooo plain three-day catgut.

cataract knife or special sclerotomy scissors. (Fig. 6.) If these measures fail the trephine may be used or a seton operation[53] may be employed.

VIII. AFTER CATARACT

Incidence. It is estimated that prior to the present trend to substitute capsulotomy forceps for the cystotome in extracting senile lenses,[54] approximately 25 per cent of cataract operations were followed by secondary cataracts of appreciable

opaqueness. Of the series of 1,004 patients, secondary membranes formed in thirty-eight patients upon whom extracapsular extraction was performed; thirty-eight discission operations were performed and it was necessary to repeat the operation in three cases. Dense secondary membranes followed five intracapsular operations. Parker[12] found that in a series of 300, secondary cataract occurred in fourteen patients

Table VII
INCIDENCE OF SECONDARY CATARACT FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Secondary Cataracts		Operation
		No.	Per Cent	
Parker[17]	150	34	22.7	Combined
	150	48	32	Simple
	150	8	5.3	Intracapsular
Parker[12]	300	30	10	Simple
		14	4.7	Combined
		3	1	Knapp-Török
Elschnig's clinic[13]	1,000	58	5.8	Capsulotomy
	44	...	....	Smith
	25	...	....	Stanculeanu
	46	...	....	Török
	76	...	....	Barraquer
Gradle[55]	100	61	61	Extracapsular
	100	6	6	Intracapsular
Berens and Bogart	302	5	1.7	Intracapsular
	702	38	5.5	Extracapsular
Total	3,145	305	9.7	

following the combined operation, in thirty after simple extraction and in three following the Knapp-Török procedure. (Table VII.)

Prevention. We agree with Gailey[18] that in performing an extracapsular cataract extraction there can be no argument concerning the use of the multiple sharp-toothed capsule forceps, taking care to remove as large a segment of the anterior capsule as is possible. If this procedure is followed it will rarely be necessary to perform a discission operation. Removal of blood and all lens matter with the aid of a Hildreth lamp assists in preventing dense secondary membranes.

Treatment. Most of these secondary cataracts are of slight or moderate thickness and respond favorably to a well-selected discission. The choice of operation for secondary cataract is important. Lyster[54] advocates freeing the iris from the secondary membrane by a sawing move-

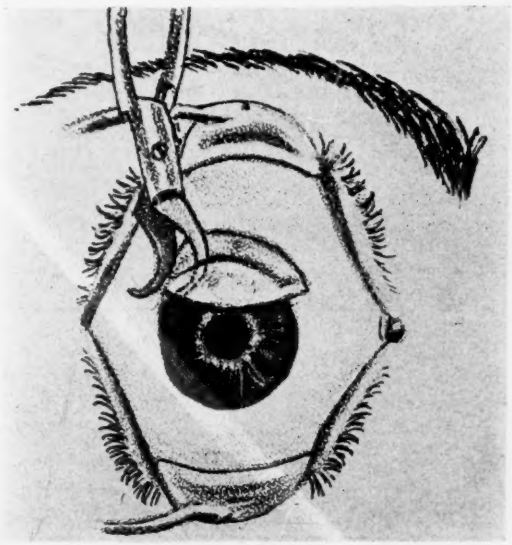


Fig. 6. Wound being reopened under a conjunctival flap with special sclerotomy scissors in a patient with secondary glaucoma.

ment with a short knife. The more experience one has, the more one is likely to agree with Fox[56] that if the membranes have any appreciable thickness they should be carefully cut with scissors. Following a discission operation retinal detachment has been observed by us in two patients and Hine[45] has also reported a case. The veil-like membranes respond well to the Ziegler[48] inverted V incision with a delicate curved knife. Dense membranes are cut with less traumatism with long thin-bladed iridocapsulotomy scissors (Fig. 7) after a small subconjunctival incision has been made at the limbus with a narrow (3 mm.) hollow ground keratome (Fig. 7A);[58] the keratome also punctures the membrane. In making these incisions, it is well to avoid the area of the wound, especially if extensive scar tissue is present. If the iris is drawn up into the wound but the pupillary border is free, a vertical incision may usually be made through the membrane (Fig. 8A and B) and the iris. When the pupillary margin is not free, it is usually better to make a horizontal incision (Fig. 9) behind the center of the cornea, using the sub-

conjunctival keratome incision and iridocapsulotomy scissors. To maintain dilatation after the anterior chamber is open in

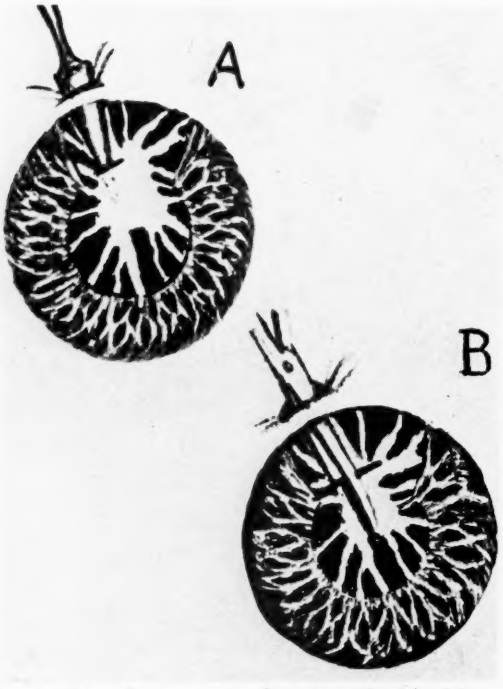


Fig. 7. Capsulotomy. A, subconjunctival incision with narrow keratome. B, cutting dense secondary membrane with long thin-bladed iridocapsulotomy scissors; pupil dilated with atropine and adrenalin.

operations for secondary membrane, atropine combined with a 1:1,000 solution of epinephrine may be used preoperatively.

IX. INTRAOCULAR INFLAMMATION

NON-SUPPURATIVE INFECTION

Subacute and Chronic Iritis and Iridocyclitis. Mild inflammation with a slight tendency to the formation of posterior synechiae is commonly observed.

Cause. Focal infections, as probable etiologic factors, have been discovered in some cases.[59] Whether highly virulent or comparatively nonpathogenic organisms and in fact what organisms or their products may cause endogenous postoperative nonsuppurative iritis and iridocyclitis, we do not know definitely[60,61] but our studies and those of Maclean[62] and Brown[63] suggest that the streptococcus is a probable etiologic factor in some cases. Traumatism may be a cause.

Traumatic iritis is more apparent,

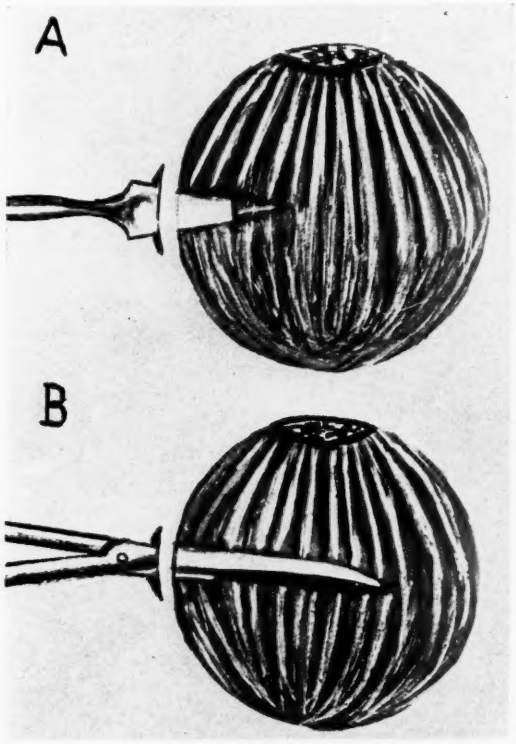


Fig. 8. A, subconjunctival introduction of keratome through limbus and then through secondary membrane and capsule. B, scissors introduced into anterior chamber, one blade passing through keratome incision in iris and capsule.

according to Mills,[6] after rough handling of the iris and after stretching and tearing of the sphincter muscle.

According to Gailey,[18] there is a greater liability to postoperative iritis and iridocyclitis among diabetic cases. In our series of patients, of the thirty-nine intracapsular cases in which diabetes was present preoperatively, iritis was not a postoperative complication. However, in the extracapsular group, two of the 109 diabetic patients developed iritis postoperatively.

Incidence. Of our series of 1,004 patients, iridocyclitis occurred in seven on whom extracapsular extraction was performed. This is a smaller percentage than we have encountered in our private patients and we believe that mild inflammations are often overlooked in hospi-

tal practice. Vision of $^{20}/_{40}$ was obtained in one patient and $^{20}/_{200}$ in two patients; the eyes of two patients were lost and final vision was not recorded in two cases. Iritis was observed in two intracapsular cases whose final vision was not recorded. Our findings and those of other ophthalmologists are tabulated in Table VIII.

Treatment. One patient with iridocyclitis, which persisted for a year in spite of all treatment and which finally involved the cornea, has been observed. After a large ethmoid cell was opened, the iridocyclitis subsided immediately and allergic reactions to all mydriatics and cycloplegics as well as to lens antigen and uveal pigment disappeared. The important relationship between bacterial products and allergy caused by non-bacterial allergens has been pointed out by Burky[64] in a study of lens protein and staphylococcus toxin. In another case, a mild iridocyclitis, which persisted for several months, was finally improved by the treatment of a severe pyorrhea, affecting two of the lower lateral incisors; when pyorrhea recurred, iridocyclitis recurred. Even though in these two cases the relationship between the focus of infection and

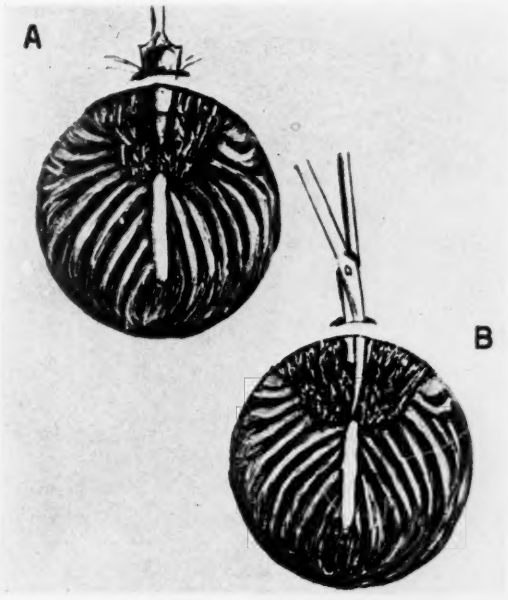


Fig. 9. A, subconjunctival incision at the limbus made with a narrow (3 mm.) hollow ground keratome. B, secondary membrane being incised with iridocapsulotomy scissors, which are passed through the secondary membrane. Vertical white line indicates where the iris may also be incised in some cases.

TABLE VIII

INCIDENCE OF IRITIS AND IRIDOCYCLITIS FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Iridocyclitis		Iritis		Operation
		No.	Per Cent	No.	Per Cent	
Bothman[10]	1,000	8	0.8	...	.....	Indian
Shipman[14]	100	8	8	...	.....	Shipman
Knapp[46]	500	35	6.6	...	.....	Intracapsular
Cruickshank[65]	113	..	....	3	3.0	Barraquer
	1,250	..	....	77	6.2	Intracapsular
Cruickshank[66]	4,027					
	3,639	..	....	...	.....	Intracapsular
	.....	..	....	153	3.79 (serous)	
	214	..	....	...	.....	Barraquer
	.....	..	....	27	0.67 (plastic)	
	174	..	....	...	.....	Capsulotomy
Appleman[4]	100	2	2	...	.....	Knapp intracapsular
Slocum[5]	882	..	....	11	1.3	Conjunctival bridge and suture
Gailey[18]	200	1	.5	...	.....	Intracapsular
	.....	2	1	...	.....	Extracapsular
Berens and Bogart	702	7	1	...	.....	Extracapsular
	302	..	....	2	0.7	Intracapsular
Total	9,176	61	0.66	273	3	

the eye lesion seems significant, it is usually difficult to determine the exact cause of an intraocular postoperative non-suppurative

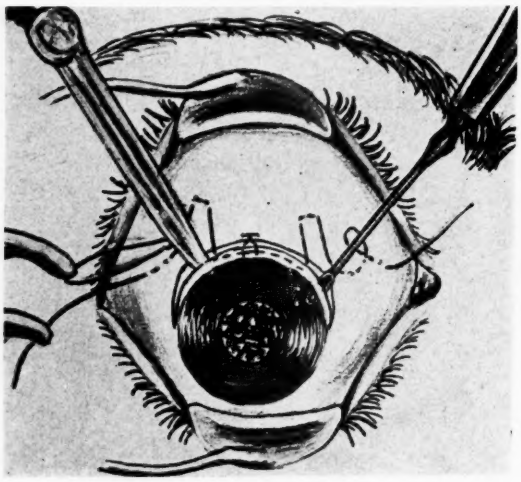


Fig. 10. Removal of retained cortex. The wound is closed with scleroconjunctival sutures; an irrigating tip is placed in one angle of the wound and a grooved spatula in the other angle.

inflammation. In New York, many patients have chronic sinusitis, especially ethmoiditis, which seems to have been an important factor in causing inflammation in a number of our patients.

Inflammation Caused by Retained Lens Substance. In some cases, toxic lens substance, that is, so-called endophthalmitis phacogenetica[67] may be a factor. Straub[68] was convinced that this type of inflammation was caused by the presence of lens matter in the eye. In other cases, there seems to be a definite sensitiveness to lens antigen, so-called endophthalmitis phacoallergica.[69] Franklin and Cordes[70] believe that this condition may be mistaken for infection.

It is especially important to perform intradermal tests (Table ix) with lens antigen in all patients who have had one eye removed, in which the lens was injured, or one eye has been operated upon for cataract, especially if marked chronic inflammation followed an extracapsular extraction. Postoperative intraocular inflammation was slight in four patients preoperatively sensitive to lens protein, who were desensitized with lens antigen alone, and in another patient, who was treated with Burky's preparation of lens antigen and staphylococcus toxin before operation, even though we were unable to remove the lens in capsule. In the light of Burky's[71] negative results in attempts to sensitize rabbits to lens antigen unless staphylococcus toxin was added to the preparation, it is important to note that three of the four patients who became hypersensitive to lens antigen had chronic sinus infection in which pathogenic staphylococci[72,73] were recovered from the sinuses. The fourth patient, after having had repeated attacks of iridocyclitis for several years, lost one eye. After enucleation a non-pathologic staphylococcus was recovered from the eye.

Prevention. If the cataract is not mature and intracapsular extraction is contraindicated, preliminary needling employing the Homer Smith[74] method is a useful procedure. After closing the wound with sutures (Fig. 10) we have had success in washing out practically all soft lens matter with half strength physiologic saline solution using the Hildreth lamp[75] to demonstrate the lens fibers. Time should be allowed for the fibers to absorb the solutions. We do not hesitate to use the

Table ix

Positive reactions to lens protein in fifty patients (91 tests) operated upon for cataract

Reaction	No. of Patients		
	Positive	Negative	Not Tested
Preoperative	28*	3	19
After first operation	43	5	11
After second operation	12		
Total	83	8	30
Number of patients desensitized	17		
Average period of desensitization	33 days		

* Ten of these patients were tested after preliminary iridectomy and in nine the lens seemed to have been injured; three patients had been operated upon for glaucoma.

spatula in the anterior chamber to dislodge adherent lens particles.

Treatment. The experience of one of us (C. B.) seems to indicate that patients may become hypersensitive to their own lens antigen. Therefore, we believe that endophthalmitis phaco-allergica may occur in spite of Rotth's[76] evidence to the contrary.

In Table IX, fifty patients (ninety-one tests), who were operated upon for cataract and who were sensitive to lens protein, are summarized. The diagnoses included in Table IX are: incipient cataract, diabetic cataract, immature cataract, traumatic cataract, secondary cataract, senile cataract, complicated cataract, Roentgen ray cataract and congenital cataract. Of this group, twenty-eight patients were sensitive to lens antigen preoperatively, three were negative and nineteen were not tested before operation. Of the twenty-eight patients, who were sensitive to lens antigen preoperatively, ten were tested after preliminary iridectomy had been performed, in nine patients the lens seemed to have been injured and three patients had been operated upon for glaucoma. Following the first operation, forty-three were positive, five were negative and eleven were not tested. Of the patients who had a second operation, twelve were positive postoperatively. Seventeen patients were desensitized for an average period of thirty-three days. The total number of reactions preoperatively was thirty-seven positive tests and five negative tests, while postoperatively there were forty-five positive tests and six negative tests.

Five patients hypersensitive to lens antigen who had lost one eye from postoperative inflammation were desensitized before the second eye was operated upon. The second operations were all successful and were followed by only slight reaction in spite of the fact that the capsule was ruptured in every case. Desensitization before operation and during the period of convalescence seems to be of some benefit.

Five patients who were not operated upon were also sensitive to lens protein. The diagnoses were diabetic cataract, complicated cataract, secondary cataract and chronic kerato-iritis, traumatic cataract and convergence insufficiency. The patient with convergence insufficiency was the mother of a boy with congenital cataracts who was sensitive to lens antigen preoperatively and lost one eye from recurrent uveitis after nine years.

Sympathetic Ophthalmitis. Sympathetic ophthalmitis, which tends to affect the second eye, is fortunately a rare complication of cataract operation.[77] According to Joy[78] sympathetic ophthalmitis occurs less frequently than its diagnosis. Therefore, a routine microscopic examination is indicated if only for statistical purposes.

Our impression is that the best treatment for these cases is the removal of or raising resistance to foci of infection, combined with the use of nonspecific therapy, especially typhoid vaccine, followed by aspiration of the anterior chamber or paracentesis. We have recently administered typhoid H antigen as suggested by Brown,[63] but are not ready to make a comparative evaluation of this antigen as compared with potent (army 58) typhoid vaccine.

In sympathetic ophthalmitis, operation to improve vision should be delayed until all signs of inflammation have been absent for at least a year. Naturally, if glaucoma occurs, it must be treated. We prefer, as a rule, to treat increased intraocular pressure in patients with sympathetic ophthalmitis and cataract by repeated aspiration (Fig. 4)[52] of the anterior chamber or paracentesis. However, in one instance, one of us (C. B.) performed iridectomy which was followed by severe inflammation with keratic precipitates; the iris became firmly adherent to the swollen lens. (Fig. 11.)

We believe that hypersensitiveness to uveal pigment or other tissue of the eye may play a rôle in the development of sympathetic ophthalmitis. (Table X.) Whether this is primary or secondary,

we do not know. Our impression is that a more important factor is the sensitization of the patient to bacteria or their products

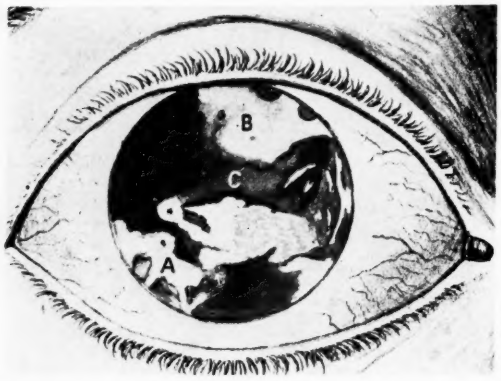


Fig. 11. Sympathetic ophthalmitis (sympathizing eye). Iris firmly adherent to the swollen lens following preliminary iridectomy. A, beginning ribbon keratitis; B, mature cataract covered by exudate; C, atrophy of the iris.

and that this is more likely to be the exciting factor which produces the more serious part of the disease.

Recently, we have followed Friedenwald's suggestion[79] of excising the skin on the fourteenth day following the injection of uveal pigment. This may be a good method of differentiating a positive from a negative or false reaction (Fig. 12) but further research is desirable. The skin lesion in Figure 12A bears some similarity to the histologic appearance of the eye usually seen in sympathetic ophthalmitis, but the eye disease, although a chronic uveitis, had none of the characteristics of sympathetic ophthalmitis and the other eye was not irritated. In Figure 12B, the negative intradermal test occurred in a case of recurrent chronic uveitis which, clinically, showed no characteristic signs of sympathetic ophthalmitis. In Figure 12C (expulsive postoperative hemorrhage with incarceration of uveal tissue in the wound) an accidental infection with abscess formation occurred and, therefore, a diagnosis could not be made from gross examination.

It is important to test these solutions for sterility as one solution of lens antigen and one of uveal pigment have recently been shown to be contaminated with staphylococci.

SUPPURATIVE POSTOPERATIVE INFECTION

Fortunately, suppurative postoperative infection is rare when rigorous methods of asepsis have been employed.

Table X
RESULTS OF INTRADERMAL UVEAL PIGMENT TESTS IN 99 PATIENTS

Diagnosis	Positive	Negative	Doubtful
Uveitis due to constitutional causes	6	18	5
Sympathetic uveitis	..	1	
Contusions with traumatic uveitis:			
No sympathetic disturbance	..	3	
With sympathetic disturbance	..	..	1
Endophthalmitis phaco-anaphylactica. (One eye lost after lens extraction)	..	..	1
Penetrating wounds involving uveal tract:			
Prolapse of iris	..	1	
Recovery without enucleation; no sympathetic ophthalmitis	..	16	1
Enucleation of injured eye; no sympathetic ophthalmitis	..	3	1
Sympathetic ophthalmitis:			
With complicated cataract (nor verified microscopically)	..	1	
Foreign body not verified pathologically	..	1	
Penetrating wound not verified pathologically	..	1	
Postoperative case, enucleation, cultures negative, pathologic report sympathetic ophthalmitis	..	1	
Acute iritis	..	1	
Recurrent iridocyclitis	..	1	
Recurrent iritis	..	1	
Chronic iridocyclitis	..	1	
Buphthalmos	..	..	1
Optic atrophy	..	1	
Cataract (postoperative iridocyclitis)*	2	14	4
Congenital cataract	..	1	
Incipient cortical cataract	..	1	
Keratitis	1	2†	
Old miliary tuberculosis of choroid	1		
Retinitis pigmentosa	1		
Macula of cornea	..	2	
Coloboma of iris	..	1	
Normal eyes	..	2	
Total of all cases subdivided	11	74	14
Total number of cases		99	

* Treated unsuccessfully with uveal pigment.
† Sympathetic ophthalmitis occurred in one case after preliminary iridectomy.

Incidence. Suppurative intraocular infection occurred in one patient of our entire series of 1,004 cases. Extracapsular extraction was performed on this patient and the eye was lost. The causative organism was not determined; silver nitrate (1 per cent)[80,81] was used preoperatively. Infections observed by us and by other surgeons have been tabulated in Table XI.

TABLE XI
INCIDENCE OF PANOPHTHALMITIS FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Panophthalmitis		Operation
		No.	Per Cent	
Bell[83]	400	2 (secondary)		
Meller[84]	249	2		
Davenport[85]	2,368	20 (suppuration)	....	Excision
Giri[23]	384	6	1.5	
Parker[17]	100	1	1	Simple
	100	1	1	Knapp-Török
	100	..	....	Combined
Parker[12]	150	1	0.66	Combined
	150	1	0.66	Simple
	150	1	0.66	Intracapsular
Beach and McAdams[15]	100	1	1	Intracapsular
Slocum[5]	682	9	1.03	
Berens and Bogart	702	1	0.14	Extracapsular
	302	..	....	Intracapsular
Total	6,137	66	0.75	

Prevention. A speculum with solid blades[82] should be used to keep the eyelashes, as well as the eyelid borders and meibomian glands, out of the field of operation. We have used 1 per cent silver nitrate as suggested by Bell.[83] The scientific basis for the employment of this drug might lie in the removal of the epithelial cells and mucous covering the conjunctiva containing pneumococci which might be carried into the eye, especially in operations in which the cornea is penetrated.[81]

Treatment. The wound should be immediately opened with a spatula as advised by Alfred von Graefe[86] and smears and cultures should be taken and prompt antiseptic treatment instituted. If any organism is found, which responds to a particular therapeutic measure, this treatment should be employed; for example, optochin in the presence of pneumococcic infection. When the cornea is invaded, the anterior chamber is usually involved; the anterior chamber may be irrigated with 1:4,000 hexylresorcinol solution. If the pneumococcus, which may be typed, is found, pneumococcus serum should be employed as suggested by McKee.[87] However, if no special organisms are found it is well to prescribe intravenous typhoid vaccine[88] or typhoid H antigen.[63]

As soon as it is apparent that the eyeball cannot be saved, a horizontal incision should be made in the cornea; the intracoular contents should be allowed to drain for a day or so or the contents may be immediately evacuated by Harold Gifford's method.[89]

X. COMPLICATIONS ASSOCIATED WITH THE LENS AND CAPSULE

RETAINED CORTEX

If a considerable amount of blood and cortex remains in the anterior chamber after operation, pain may result from irritation of the tissues by toxins or from an allergic reaction in others. Knapp[90] reports that retained cortex was present in two cases in a series of 200 capsulotomy cataract operations.

Prevention. The importance of desensitizing injections of lens antigen in certain cases has been discussed under postoperative inflammation.

Treatment. Banaji[91] recommends the injection of 10 cc. of boiled milk immediately after operation if a large amount of lens matter is retained in the anterior chamber. It is claimed[92] that diathermy may restore partial vision in some patients with senile cataract. Although we have had no experience with diathermy in the treatment of cataract, it may be a valuable adjunct to the use of dionin and hot compresses by promoting more rapid absorption in patients with retained cortex.

XI. LOSS OF VITREOUS

Cause. Subchoroidal hemorrhage may be the exciting factor in the loss of vitreous, but injury to the eye, vomiting, straining or traumatism are more common causes. It is stated[93] that tying of sutures involves a real risk, for in the short period of time necessary for this procedure, a considerable amount of vitreous may be lost. However, this criticism does not seem to apply to the untied suture and conjunctival bridge.[94]

Incidence. Loss of vitreous occurred in thirty of the 302 intracapsular extractions

(9.9 per cent), whereas this complication was observed in sixty-three (9.0 per cent) of the extracapsular extractions. These figures

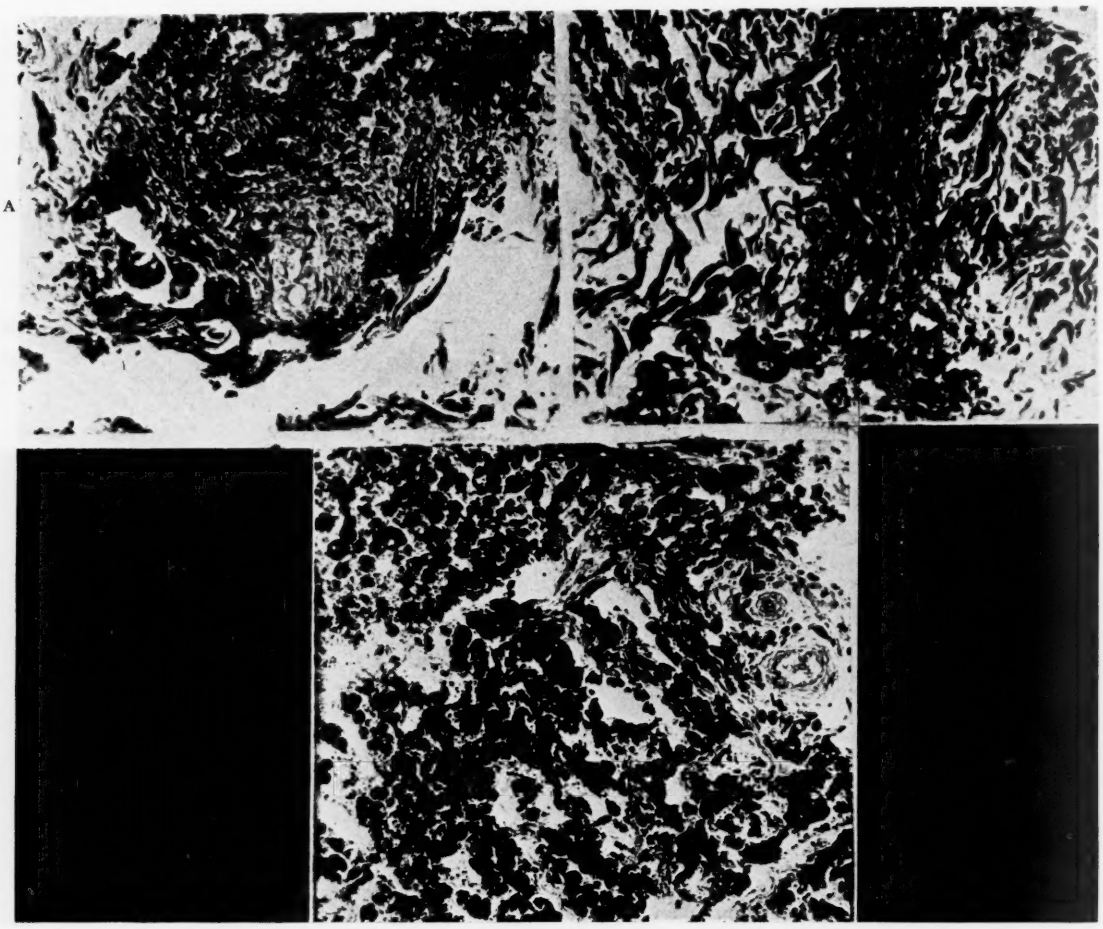


FIG. 12. Histologic picture of intradermal reactions to uveal pigment. A, positive intradermal reactions to uveal pigment; B, negative intradermal reaction to uveal pigment; C, accidental infection with abscess formation following intradermal tests with uveal pigment.

possibly are significant in regard to the greater danger from vitreous loss following intracapsular extraction in the hands of the average surgeon, when we realize that extraction in capsule usually was not attempted in patients with complicated cataracts. (Table XII.) Greater freedom from postoperative inflammatory complications and the necessity for the dangerous operation of discission must be considered in evaluating intracapsular and extracapsular extractions.

Prevention. A wound thoroughly closed, as soon as the lens is expressed, by a complete conjunctival flap, held by sutures, which include the episcleral tissue, and the presence of the iris are safe-guards. Careful nursing to see that the patient avoids strain or injury is important. Coughing should be controlled by sedatives, posture and local nasal treatments. Vomiting caused by drugs should be prevented by preoperative trial of the drugs to be employed postoperatively. If cataract is present in only one eye, the vitreous in this eye is usually liquid. A flap should be prepared and sutures should be in place so that the lens may be extracted with a spoon.

Treatment. If much solid vitreous is incarcerated and the conjunctival flap is not wide and complete, excision of the protruding vitreous is indicated after carefully anesthetizing the eye. Before excising

the vitreous, the conjunctiva above the cornea should be undermined and two

TABLE XII
INCIDENCE OF LOSS OF VITREOUS FOLLOWING CATARACT EXTRACTION

Surgeon	No. of Cases	Loss of Vitreous		Operation
		No.	Per Cent	
Bothman[10]	1,000	45	4.5	Indian
Dunphy[11]	1,821	130	7.1	Combined
	298	2	0.67	Simple
	441	83	18.8	Intracapsular
Parker[36]	1,013	109	10.7	Combined
	156	10	6.4	Simple
	49	5	10.2	Knapp
	91	18	19.7	Indian
	104	10	9.6	Preliminary iridectomy
Ellett[93]	600	52	7.6	
Elschnig's Clinic[13]	1,000	19	1.9	Capsulotomy
	44	9	20	Smith
	35	9	11	Stanculeanu
	48	4	9	Török
	78	8	10	Barraquer
Parker[12]	300	4	.....	Combined
		2	4.0	Simple
		6	.....	Knapp-Török
Shipman[14]	100	.....	.....	Shipman
Knapp[46]	500	42	8.4	Intracapsular
Grosz[96]	15,050	376	2.5	1 per cent extracapsular. 4 per cent intracapsular
Greenwood and Grossman[97]	1,343	83	6.2	Intracapsular
Parker[17]	150	3	2	Combined
	150	5	3.3	Simple
	150	9	6	Knapp-Török
Townes[98]	73	6	8.3	Intracapsular
Gailey[18]	200	9	4.5	Extracapsular
		4	2	Intracapsular
Berens and Bogart	702	63	9	Extracapsular
	302	30	9.9	Intracapsular
Total	25,794	1,153	4.48	

double-armed sutures placed so that the flap may be made to cover the cornea immediately. If a good conjunctival flap is present sutures should be placed in this.

In a patient seen by one of us (C. B.), a large vitreous prolapse incompletely covered by conjunctiva caused no serious difficulty and operation finally resulted in a well-healed wound and $^{20}/_{20}$ vision.

XII. POSTOPERATIVE MENTAL DISORDERS

Postoperative mental disorders may be classified as: (1) exhaustion psychoses; (2) toxic psychoses; (3) delirium in alcoholic patients; (4) mental confusion in senility; and (5) hysterical amblyopia. In old people, especially alcoholic patients, confinement in bed with their eyes bandaged causes insomnia, irritation and, at times, true delirium. The state of agitation may be severe and the patient may remove the dressing or even get out of bed.

Cause. We agree with certain surgeons[99,100,101] who believe that prolonged bandaging of both eyes is the most important single causative factor. This assumption seems to be strengthened by the fact that in the last 152 operations on private patients performed by one of us (C. B.) in which the patients were permitted to use one eye, either at the time of operation or within two days, no marked mental disturbances developed. Two of the patients had definite psychologic difficulties and were under the care of a neurologist at the time of operation.

It is our impression that toxic causes may be important, especially toxemia caused by hypersensitiveness to certain sedative drugs and particularly cycloplegics like atropine. We use atropine sparingly, unless definitely indicated, and always apply an ointment to the inner canthus before instilling the drug. Because intestinal toxemia may be a factor in some cases, we often prescribe colonic irrigations the

TABLE XIII
INCIDENCE OF DELIRIUM FOLLOWING CATARACT EXTRACTION

Surgeon	Number of Cases	Delirium		Operation
		No.	Per Cent	
Parker[36]	1,421	33	2.3	
Harbridge[104]	7*	5	7.2	
Parker[12]	100	3	3	Combined
	100	1	1	Knapp-Török
	100	..	....	Simple
Berens and Bogart	302	1	0.33	Intracapsular
	702	1	0.14	Extracapsular
Berens†	152	..	....	Intracapsular
				Extracapsular
Totals	2,884	44	1.53	

* These patients were all over 80 years of age.
† Patients given a Ring's mask with a hole before one eye at the time of operation.

next day and when indicated. An hysterical tendency may result in hysterical amblyopia.[102]

Incidence. Greenwood[101] estimates that some degree of mental disturbance develops in from 2.5 to 3 per cent of all cases of cataract. In Table XIII the occurrence of delirium following cataract extraction by various surgeons is tabulated. In our series of 1,004 cases, mental disturbance occurred in two patients.

Prevention. Elderly patients should be carefully watched during the first two or three days and nights after operation. If possible, sutures should be employed and the patient permitted to sit up in bed the same day if he feels strong enough, and allowed out of bed the day after the operation.

These patients should be permitted to use one eye at the time of operation, by making a hole (15 mm. in diameter) in the Ring's mask directly in front of one eye. Because preventive measures should begin before operation, the preoperative study of a patient's mental condition is important. Especial effort should be made to gain the patient's confidence. Some patients should be told of the nature of cataract and be informed of the various procedures to be followed, stressing freedom from pain. The use of mild sedatives for several days before operation is of value.

Treatment. When delirium has developed, one eye should be uncovered and large doses of sedatives administered. The patient should sit up in bed or in a chair; a person to whom he is devoted should be with him. Scopolamine may be administered in obstinate cases but variability of the action of this drug renders it less valuable than others.[103] The most useful sedatives are the bromides and chloral. One or the other of these may be badly tolerated or ineffectual in some patients and other drugs may have a particularly beneficial action. In alcoholic patients, it is usually wise to administer the amount of alcohol to which they have become habituated.

XIII. POSTOPERATIVE PNEUMONIA AND BRONCHITIS

Elderly patients should not be kept on their backs for too long a time because there is a tendency for them to develop pneumonia, bronchitis and other pulmonary complications. For this reason, the wound should be completely closed with sutures and the patient permitted to sit up in bed and allowed to be out of bed as soon as possible.

Incidence. In our series of 1,004 cases pneumonia occurred in only one patient on whom extracapsular extraction was performed.

POSTOPERATIVE PAIN

Rupture of the wound is a common cause of postoperative pain. In these cases, the eyes may have been free from pain for twenty-four hours after operation. However, when the dressing is removed they may be saturated with tears and when the eyelids are separated, watery fluid which is free of mucus or pus may escape.

In other cases the pain is caused by prolapse of the iris and possibly by movements of the hypersensitive tissues or traction of a suture. Other causes of postoperative pain may be postoperative glaucoma, hemorrhage,[22] postoperative iritis and dressing which irritate the cornea because of incomplete closure of the eyelids.

XIV. SUMMARY

Many complications following cataract operations frequently result in partial or complete loss of visual acuity. Therefore, the prevention and treatment of these complications is of vital importance.

One thousand and four clinic patients were operated upon at the New York Eye and Ear Infirmary during the last two years. Extracapsular extractions were performed on 702 patients and intracapsular extractions on 302.

The most frequent postoperative complications were: hemorrhage into the anterior chamber (4 per cent in the intra-

capsular cases and 1.4 per cent in the extracapsular cases), and loss of vitreous (9.9 per cent in the intracapsular cases and 9.0 per cent in the extracapsular cases). The other complications, for example, detachment of the retina, detachment of the choroid, suppurative intraocular infection, and prolapse of the iris, occurred in only a few cases of each series. Secondary glaucoma occurred in seven extracapsular cases; iridocyclitis was observed following seven extracapsular extractions and two intracapsular operations.

If the number of postoperative complications is to be reduced, there seems to be little question that the preliminary examination is most important. At this examination, urine, intraocular tension, blood pressure, the Wassermann reaction, blood sugar, blood urea, basal metabolism, Roentgen rays of the sinuses and teeth, cardiovascular disease, bronchitis, and diabetes should be given special consideration. Coagulation, bleeding time and the Schilling index should be known, in addition to the usual blood count and hemoglobin estimation. In taking smears and cultures of the conjunctival sac, Elschnig's[3] method of using blood serum to extract the bacterial flora for the culture is excellent. Examination should be made of the gums, teeth, sinuses, throat, tonsils and lacrimal sac. If abscessed teeth, infection of the lacrimal sac, sinus infection, increased intraocular tension or increased blood pressure are found, these conditions should be treated before operation. It is also important to know whether the pupil can be properly dilated. Gailey[18] recommends the instillation of euphthalmine for this purpose. Careful nursing, early treatment of complications, and abstinence from unnecessary examinations and manipulations are the most important factors in postoperative care. However, daily dressings are advisable in order to detect complications in their incipiency and to allay the patient's apprehension.

Complications following cataract extractions may often be prevented by attention to the following details:

1. Careful preoperative general physical study of the patient; determination of the effects of sedative drugs; blood studies (hemoglobin, Schilling index and sedimentation rate); Wassermann test; Mantoux test; and gonorrheal fixation test.
2. Normal coagulation and bleeding time.
3. Treatment of syphilis and control of diabetes and high blood pressure.
4. Study of patient's sensitization to lens antigen and uveal pigment preoperatively, if one eye has been operated upon, especially if postoperative inflammation developed.
5. Preoperative elimination of foci of infection or raising resistance to them.
6. A preoperative study of the patient's psychology in order to allay fear.
7. Employment of a retrobulbar injection of procaine and adrenalin in preparing for operation.
8. Instillation of 1 per cent silver nitrate and argyrol preoperatively to prevent suppurative postoperative infection.
9. Adequate anesthesia and akinesia.
10. Care in employing and selecting instruments.
11. Lifting the speculum away from the eyeball when the lens is being expressed.
12. Exercise of care in using capsule forceps and the cystotome in order to avoid dislocating the lens.
13. A sufficiently large section.
14. Care in making the incision to obtain a complete conjunctival flap.
15. One-half strength physiologic saline solution warmed to body temperature and used to remove all retained cortex under ultraviolet rays.
16. When sutures are not employed, the flap must be in proper position.
17. Complete closure of the wound by sutures as soon as the lens presents and pinching together of the lips of the conjunctival wound at the completion of the operation.
18. Proper care of the patient during and between dressings.
19. Patient should not be permitted to strain or cough.

20. Gradual light adaptation of eyes during dressings.

21. Avoidance of observation of the upper border of wound or touching upper eyelid. The patient should be told what you are about to do.

22. Avoidance of strong miotics after primary instillation, unless prolapse of iris occurs.

23. Careful toilet of the wound.

24. To prevent postoperative mental disorders, elderly patients should be carefully watched during the first two or three days and nights after operation, and should have a hole cut in Ring's mask at the primary dressing.

25. The patient should be allowed to sit up in bed the same day if possible and to be out of bed the day after operation.

26. To prevent operative and postoperative loss of vitreous the wound should be thoroughly closed by a complete conjunctival flap held by sutures which include the episcleral tissue.

The following treatment of postoperative complications of cataract extraction may be considered:

1. For abrasion of the cornea and tendency for the iris or vitreous to prolapse, a firm dressing should be applied.

2. For delayed wound closure, iridectomy or excision of vitreous or clearing the wound in other ways after preparation of a conjunctival flap.

3. For striate keratitis, hot applications and instillation of dionin.

4. For cystoid scars and small beads of prolapsed iris, application of trichloracetic acid.

5. For extensive prolapse of the iris not covered by conjunctiva, a small iridectomy may be performed and the wound closed with sutures.

6. For extreme prolapse of the iris, a conjunctival flap may be made by dissecting from the cornea upward.

7. For repeated hemorrhages, calcium gluconate intravenously, normal horse serum, snake venom and brain extract may be administered.

8. For detachment of the choroid, the patient should rest with head elevated, and measures to increase coagulation of blood and sedatives should be given.

9. For hypertension caused by iridocyclitis, aspiration of the anterior chamber or paracentesis and mydriatics.

10. For persistent hypertension, iriodcorneosclerectomy following simple extraction; if an iridectomy has been performed, corneosclerectomy over one pillar combined with iridencleisis may be employed.

11. For postoperative non-suppurative inflammation, examination for and treatment of chronic infections.

12. For iritis caused by retention of protein in the anterior chamber, atropine, infra-red rays, autohemic injections and typhoid vaccines or other foreign proteins.

13. For endophthalmitis phaco-allergica, desensitization to lens antigen and staphylococcus toxin before operation and during convalescence if the patient is sensitive to lens antigen.

14. Desensitizing injections of uveal pigment may be used if hypersensitiveness to uveal pigment develops coincidently with postoperative inflammation.

15. For suppurative infection, smears and cultures (pneumococcus serum, P.R.N.); anterior chamber should be washed out with 1:4,000 hexylresorcinol solution; intravenous typhoid vaccine or typhoid "H" antigen should be given.

16. For delirium, sedatives; patient should sit up in bed or in a chair; scopolamine in obstinate cases.

17. For expulsive hemorrhage, sedatives, sutures, scleral puncture and lightly compressing dressing are necessary.

XV. CONCLUSIONS

The following conclusions may be justified:

1. Careful preoperative study of our patients, combined with the institution of treatment for chronic infections, hyperglycemia, hypertension and other general diseases, may prevent serious postoperative

complications which frequently result in impaired vision or blindness.

2. It is important to test the reaction of patients to lens antigen if one eye has been operated upon for cataract, and if the extracapsular method has been employed. If the lens antigen test is positive, desensitizing injections of lens antigen and staphylococcus toxin should be administered, especially if the operation was followed by inflammation. If hypersensitiveness to lens antigen develops postoperatively, desensitization with lens antigen and staphylococcus toxin seems to be of value.

3. If patients have had severe uveitis in one eye, if one eye has been enucleated for sympathetic ophthalmitis or postoperative inflammation develops, intradermal tests with uveal pigment with the later excision and microscopic examination of skin should be practiced. If the test is positive, desensitizing injection of uveal pigment may be administered. Chronic infections should be treated.

4. Study of a series of 1,004 cataract operations performed by various surgeons, many by house surgeons, seems to indicate that postoperative complications are about equally distributed between the intracapsular and extracapsular operations.

5. Of the 1,004 operations, 30 per cent were intracapsular extractions. The number of immediate and postoperative complications was approximately the same in the two groups. This suggests that, at least at the New York Eye and Ear Infirmary, the intracapsular extraction is a valuable and a fairly safe procedure in the hands of the average surgeon. It must be remembered that 17 per cent of the extracapsular cases were attempted intracapsular extractions.

6. Careful complete suturing of the wound with a complete but not too wide conjunctival flap seems to be one of the best methods of preventing many serious postoperative complications.

7. In the postoperative care of the patient, the most important single factor in preventing complications, other than careful nursing, the treatment of cough as well as other movements which might endanger the eye, is the careful postoperative examination of the eye, especially avoiding squeezing by unnecessary or clumsy manipulation.

Finally, it would seem that thorough preoperative examination and treatment combined with proper preparation, skilful manipulation and complete suturing with a conjunctival flap during the operation, followed by judicious postoperative observation, nursing and treatment will prevent or render comparatively harmless a great many postoperative complications which often result in partial or complete blindness.

We wish to acknowledge the courtesy of the following ophthalmologists in allowing us to include a number of their cases in this report: Dr. Francis W. Shine, Dr. Webb W. Weeks, Dr. Bernard Samuels, Dr. Clyde F. McDannald and Dr. Ben Witt Key.

We are also grateful to the staff of the Record Room at the New York Eye and Ear Infirmary for their coöperation in compiling these statistics.

REFERENCES

1. Cruickshank, M. M. Choroidal hemorrhage following cataract extraction. *Indian M. Gaz.*, 60: 213, 1925.
2. Nugent, C. B. Complications in cataract extraction. *Am. J. Opbth.*, 17: 135, 1934.
3. Elschnig, A. Die intrakapsuläre Starextraktion. Berlin, 1932. J. Springer.
4. Appleman, L. F. Intracapsular cataract extractions by the Knapp method: a report on one hundred cases. *Arch. Opbth.*, 9: 907, 1933.
5. Slocum, G. Employment of a conjunctival bridge and suture in cataract extraction. *Arch. Opbth.*, 10: 329, 1933.
6. Mills, L. Iritis, iridocyclitis and prolapse of the iris in modern surgery. *Tr. Sect. Opbth., A. M. A.* 1928, p. 185.
7. Pallares Lluesma, J. Hernia del iris, complicación de la operación de catarata en ojos con pterigion. *Arch. de oftal. bispano-am.*, 35: 326, 1935.
8. Bulson, A. E. Prolapse of iris after cataract extraction. *Tr. Sect. Opbth., A. M. A.*, 1925, p. 69.
9. Gala, A. Prispevky k leceni postoperativnich komplikaci fo extrakci katarakt. *Bratisl. lekar. listy.*, 11: 11, 1931.
10. Bothman, L. Complications of cataract surgery in India. *Illinois M. J.*, 62: 243, 1932.
11. Dunphy, E. B. Loss of vitreous in cataract extraction. *Tr. Sect. Opbth., A. M. A.*, 1927, p. 229.
12. Parker, W. R. Cataract extraction, comparative results obtained by the combined, simple and Knapp-Török methods of procedure. *J. A. M. A.*, 89: 2252, 1927.

13. Elschnig, A. Extraction of senile cataract in the capsule. *Am. J. Opbth.*, 8: 355, 1925.
14. Shipman, J. S. A safe method of cataract extraction: with report of results in 100 operations. *J. M. Soc. New Jersey*, 29: 852, 1932.
15. Beach, S. J., and McAdams, W. R. Intracapsular extraction of cataract in the average practice. *Arch. Opbth.*, 15: 95, 1936.
16. Verhoeff, F. H. A corneo-sclero-conjunctival suture in operations for cataract. *Tr. Am. Opbth. Soc.*, 25: 48, 1927.
17. Parker, W. R. Comparative results in the extraction of senile cataracts. *Arch. Opbth.*, 11: 183, 1934.
18. Gailey, W. W. A report of two hundred cataract operations. *Illinois M. J.*, 72: 528 (Dec.) 1937.
19. Bettman, J. W., and Barkan, H. Trichloracetic acid in the treatment of iris prolapse. *Am. J. Opbth.*, 20: 131, 1937.
20. Greeves, A. Implantation cyst following cataract extraction. *Roy. Soc. Med.*, London, Jan. 8, 1932; *Am. J. Opbth.*, 15: 851, 1932.
21. Krug, E. F. A cyst of the posterior chamber with a microscopic study of the eye. *Am. J. Opbth.*, 21: 413, 1938.
22. Vail, D. T., Jr. On hyphema after cataract extraction. *Tr. Am. Opbth. Soc.*, 31: 496, 1933.
23. Giri, D. V. Some observations on intracapsular extraction of cataract, with description of a simple technique. *Tr. Opbth. Soc. United Kingdom*, 43: 248, 1923.
24. Black, M. Hemorrhage after cataract operation. *Am. J. Opbth.*, 7: 539, 1934.
25. Thilliez, Professor. Un traitement des hémorrhagies expulsives consécutives à l'extraction de la cataracte. *Soc. franç. d'opbt.*, 38: 406, 1922.
26. Abramowics, I. Introduction of air into the anterior chamber in the course of cataract operation. *Ann. d'ocul.*, 167: 581, 1930.
27. O'Brien, C. S. Detachment of the choroid after cataract extraction. *Arch. Opbth.*, 14: 527, 1935.
28. Hill, H. F. Personal communication. April 1, 1938.
29. Fuchs, E. Ablösung der Aderhaut nach Staroperation. *Arch. f. Opbth.*, 51: 199, 1900.
30. Barkan, H. Postoperative detachment of choroid: special reference to Elliot's operation for glaucoma. *Tr. Sect. Opbth., A. M. A.*, 1915, p. 209.
31. Jensen, V. A. Hemorrhages into anterior chamber of the eye in cataract operations with and without employment of adrenalin. *Acta Opbth.*, 10: 382, 1932.
32. Manes, A. J. Experiences in 280 intracapsular extractions. *Arch. de oftal. de Buenos Aires*, 5: 382, 1935.
33. Clapp, C. A. Surgical intervention for cataract. *Arch. Opbth.*, 18: 827, 1937.
34. Gifford, S. R. The prevention of complications in the cataract operation. *Illinois M. J.*, 68: 243, 1935.
35. Gros, H., and Fromaget, H. Deux cas d'hémorragies sous-choroïdiennes expulsives au cours de l'opération de la cataracte. *Ann. d'ocul.*, 153: 476, 1916.
36. Parker, W. R. Senile cataract extraction: a comparative study of results obtained in 1,421 operations. *Tr. Sect. Opbth., A. M. A.*, 1921, p. 202.
37. Barraquer, I. Phakoerisis. *Am. J. Opbth.*, 3: 721, 1920.
38. Wolfe, O. Summary of one hundred Barraquer cataract extractions. *Am. J. Opbth.*, 18: 556, 1935.
39. Verhoeff, F. H. Scleral puncture of expulsive subchoroidal hemorrhage after sclerotomy. *Opbth. Rec.*, 24: 55, 1915.
40. Woodruff, H. W. Management of complications in the operation for senile cataract. *Am. J. Opbth.*, 19: 146, 1936.
41. Ziegler, S. L. History of discission of the lens, with the technique of complete discission by the V-shaped method. *Tr. Opbth. Soc. United Kingdom*, 45: 154, 1925.
42. Vail, D. Posterior sclerotomy as a form of treatment in subchoroidal expulsive hemorrhage. *Tr. Am. Opbth. Soc.*, 35: 343, 1937.
43. Shapland, C. D. Retinal detachment and its treatment by surgical methods. *Brit. J. Opbth.*, 18: 1, 1934.
44. Cruise, R. Detachment following simple extraction of cataract. *Arch. Opbth.*, 15: 576, 1936.
45. Hine, M. L. Reattachment of the retina after discission of soft cataract. *Roy. Soc. Med.*, London; *Am. J. Opbth.*, 18: 558, 1935.
46. Knapp, A. Complications of forceps intracapsular operation for cataract based on analysis of 500 successive cases. *Arch. Opbth.*, 16: 770, 1936.
47. Peter, L. C. Intracapsular cataract extraction: the relative merits of the modern methods and indications for their use. *Tr. Am. Acad. Opbth.*, 1933, p. 99.
48. Berens, C., and Sitchevska, O. Complete discission of the crystalline lens. *Tr. Am. Acad. Opbth.*, 1932, p. 283.
49. Ferrer, H. Tonometry before and after cataract operation. *Am. J. Opbth.*, 15: 324, 1932.
50. Berens, C. Iridocorneosclerectomy for glaucoma. *Am. J. Opbth.*, 19: 470, 1936.
51. Fox, S. A. Postoperative glaucoma. *Arch. Opbth.*, 16: 585, 1936.
52. Berens, C. Aspiration for Glaucoma. *Bull. O. O. R. L. Soc., Kansas City*, 7: 5 (Feb.) 1938.
53. Wolfe, O. R., and Blaess, M. J. Seton operation in glaucoma. *Am. J. Opbth.*, 19: 400, 1936.
54. Lyster, T. C. Management of dense secondary cataract. *Am. J. Opbth.*, 16: 122, 1933.
55. Gradle, H. Intracapsular versus extracapsular extraction of cataract. *Tr. Sect. Opbth., A. M. A.*, 1933, p. 186.
56. Fox, L. W. Causes of failure in ophthalmic operations. *Arch. Opbth.*, 3: 695, 1930.
57. Berens, C. Iridocapsulotomy scissors with new features. *J. A. M. A.*, 87: 1301, 1926.
58. Berens, C. A new keratome: a hollow-ground narrow angular keratome for paracentesis, iridocapsulotomy and iridotomy. *Tr. Am. Acad. Opbth.*, 1937, p. 485.
59. Gifford, S. A review of the literature on the etiology of acute iritis. *Tr. Assn. Research Opbth.*, 1930, p. 1.
60. Berens, C., Connolly, P. T., and Chapman, G. H. Focal infection in diseases of the eye; 1. Report of certain laboratory examinations. *Brit. J. Opbth.*, 18: 463, 1934.

61. Berens, C., Nilson, E. L., and Chapman, G. H. Iritis produced in rabbits' eyes by the intravenous injection of crude and purified cultures of bacteria isolated from patients with certain inflammatory eye diseases. *Am. J. Ophth.*, 19: 1060, 1936.
62. MacLean, A. L. Experimental iritis: the ocular reactions in rabbits sensitized to streptococcus viridans. *Tr. Am. Ophth. Soc.*, 32: 324, 1936.
63. Brown, A. L., and Pugh, J. Effect of intraocular concentration of typhoid antibodies on experimental corneal ulcers, with report of 7 cases. *Arch. Ophth.*, 16: 476, 1936.
64. Burky, E. L. Studies on the action of staphylococcus toxin and antitoxin with special reference to ophthalmology. *Am. J. Ophth.*, 19: 841, 1936.
65. Cruickshank, M. M. Iritis following cataract. *Indian M. Gaz.*, 61: 490, 1926.
66. Cruickshank, M. M. Complications following 4000 cases of cataract extraction. *Brit. J. Ophth.*, 11: 275, 1927.
67. Gifford, S. R. Allergic and toxic properties of lens protein. *Tr. Sect. Ophth., A. M. A.*, 1925, p. 82.
68. Straub, M. Over Outslekingen door Oplossing Van Lensmassa. Amsterdam, 1919.
69. Verhoeff, F. H., and Lemoine, A. N. Endophthalmitis phacoanaphylactica. *Tr. Internat. Cong. Ophth.*, Washington, D. C., 1922, p. 234.
70. Franklin, W. S., and Cordes, F. C. Postoperative cataract infections. *J. A. M. A.*, 91: 1977, 1928.
71. Burky, E. L., and Henton, H. C. Staphylococcus toxin combined with lens extract as a desensitizing agent in individuals with a cutaneous sensitivity to lens extract. *Am. J. Ophth.*, 19: 782, 1936.
72. Chapman, G., Lieb, C., and Curcio, L. The use of bromthymol blue agar and phenol red manitol agar for the isolation of pathogenic types of staphylococci. *J. Clin. Path. (Tech. Sup.)*, 2: 3, 1938.
73. Chapman, G., Berens, C., Nilson, E. L., and Curcio, L. Differentiation of pathogenic staphylococci from nonpathogenic types. *J. Bact.*, 35: 311, 1938.
74. Smith, Homer E. Safe and speedy extraction of the immature cataractous lens following preliminary iridectomy. *Tr. Sect. Ophth., A. M. A.*, 1914, p. 177.
75. Hildreth, H. R. A surgical ultraviolet lamp as an aid to the removal of dislocated lenses. *Am. J. Ophth.*, 17: 414, 1934.
76. Rotth, A. On the question of phacoanaphylactic endophthalmitis. *Arch. Ophth.*, 55: 103, 1926.
77. Berens, C. Discussion of Bedell's paper on "Clinical manifestations of allergy in the eyes." *New York State J. Med.*, 36: 959 (July 1) 1936.
78. Joy, H. H. Sympathetic ophthalmitis following intraocular operations. *New York State J. Med.*, 36: 1498, 1936.
79. Friedenwald, J. S. Notes on the allergy theory of sympathetic ophthalmitis. *Tr. Assn. Research Ophth.*, 1934, p. 47.
80. Bell, G. H. Prevention of postoperative infections. *Tr. Sect. Ophth., A. M. A.*, 1921, p. 217.
81. Berens, C. Discussion of Bell's paper on prevention of postoperative infections. *Tr. Am. Ophth. Soc.*, Vol. 25, 1925.
82. Berens, C. An eye speculum. *Tr. Sect. Ophth., A. M. A.*, 1932, p. 357.
83. Bell, G. H. New method of preventing postoperative intraocular infections. *Tr. Sect. Ophth., A. M. A.*, 1921, p. 217.
84. Meller, J. In Hans Barkan: Cataract extractions performed by Prof. Josef Meller of Vienna 1919–1921. *J. A. M. A.*, 81: 2086, 1923.
85. Davenport, R. C. The after results of cataract extraction. *Brit. J. Ophth.*, 12: 85, 1928.
86. Von Graefe, A. Quoted by de Wecker, L. Ocular Therapeutics. London, 1879. Smith Elder Co., p. 333.
87. McKee, S. H. A study of the pneumococcus group from the inflamed conjunctiva and lacrimal sac. *Am. J. Ophth.*, 18: 1021, 1935.
88. Berens, C., Losey, R. R., Hardy, L. H., and Meek, R. E. Studies in immunology in relation to ophthalmology; first communication. *Atlantic M. J.*, 30: 547, 1927.
89. Gifford, H. On strictly simple evisceration of the eyeball. *Arch. Ophth.*, 29: 422, 1900; On the technique of evisceration. *Arch. Ophth.*, 44: 162, 1915.
90. Knapp, A. Certain debatable steps in the operation for senile cataract. *Tr. Am. Acad. Ophth.*, 1928, p. 132.
91. Banaji, B. P. Avoidance of postoperative infection in cataract operations. *Brit. J. Ophth.*, 11: 75, 1927.
92. Alloway, F. L., and Funk, R. S. Diathermy in senile cataract. *Illinois M. J.*, 55: 286, 1929.
93. Greens' Eye Hospital Bulletin of Practical Ophthalmology. Warning in the use of dinitrophenol, 5: 59 (Oct.) 1935.
94. Berens, C., and Losey, R. R. An untied conjunctival suture and conjunctival bridge in cataract extractions; results and conclusions. *Brit. J. Ophth.*, 10: 577, 1926.
95. Ellett, E. C. Loss of vitreous in cataract extraction. *Tr. Sect. Ophth., A. M. A.*, 1928, p. 223.
96. Grosz, E. de. Cataract extraction in 15,000 operations. *Arch. d'opht.*, 53: 161, 1936.
97. Greenwood, A., and Grossman, H. P. Analysis of 1,343 intracapsular cataract extractions by 48 operators following verhoeff method. *Tr. Am. Ophth. Soc.*, 33: 353, 1935.
98. Townes, C. D. One hundred attempted intracapsular operations. *Kentucky State M. J.*, 34: 444, 1936.
99. Parker, W. R. Post-cataract extraction delirium. *Tr. Sect. Ophth., A. M. A.*, 1913, p. 395.
100. Posey, W. C. Mental disturbances after operations upon the eye. *Ophth. Rev.*, 19: 233, 1900.
101. Greenwood, A. Mental disturbances following operations for cataract. *Tr. Sect. Ophth., A. M. A.*, 1928, p. 233.
102. Garcia, D. S. Cataract compliquée d'amaurose hystérique. *Clin. opht.*, 22: 333, 1917.
103. Thomas, F. C. Delirium following cataract operations. *Kentucky State M. J.*, 24: 134, 1928.
104. Harbridge, D. F. Senile cataract extraction followed by certain complications. *Ann. Ophth.*, 23: 58, 1914.

MANAGEMENT OF THE COMPLICATIONS OF INTRAOCULAR SURGERY

EVERETT L. GOAR, M.D., F.A.C.S.

Ophthalmologist Hermann, Southern Pacific and Houston Eye, Ear, Nose and Throat Hospitals

HOUSTON, TEXAS

ANY discussion of the complications of intraocular surgery must naturally fall into two groups—prevention of such complications and treatment after they occur. Many complications may be avoided by the systematic employment of methods that diminish the hazards of intraocular surgery. The scope of this paper will not permit discussion of all complications that may happen in every intraocular operation, and as the purpose of the article is to present the subject from the most practical standpoint possible, it will be confined largely to the two conditions that every ophthalmic surgeon must attempt to alleviate, viz., cataract and glaucoma.

The bête noire of all ophthalmic surgeons, as with general surgeons, is hemorrhage and infection, and this may occur at the time of operation or later during convalescence. If these two hazards could be eliminated, the life of the ophthalmic surgeon would be much more carefree than it is now.

Many of the details in the technique of intraocular surgery which could diminish the hazards of complications are not used even by the best ophthalmic surgeons. Some operators are so skilful with their hands and have such complete control of their patients that they do not feel the need of safety devices. Others do not agree that such methods diminish the hazards, and as one watches surgeons in various clinics and studies their technique, it becomes apparent that to a large extent, the results of eye surgery depend upon the surgeon, and each surgeon must develop the technique that is safest in his hands. No procedure has been devised to prevent complications in the hands of an untrained or heavy-handed surgeon. This paper is therefore written by an average ophthalmic surgeon for others of the same type, and only methods that have been put to the test of actual experience will be advocated.

CATARACTS

When a patient consults the surgeon as a prospective candidate for cataract operation, there are certain essential facts that must be ascertained. A rubber stamp containing the following data is used on the record:

Pre Op. Cataract Record	
V. R.	L.
Lt. Proj.	
Color	
Lacrimal system	
Tension R.	L.
Stage	
Smear	
Culture	
B. P.	
Urine	
Focal infection	
Bleeding time	
Clotting time	

Each of the subheads bears a direct relation to the prevention of a complication during or following operation. Each of these will be briefly discussed.

The vision of each eye is an important bit of evidence to have upon the history, and vision with the best correcting lens should be recorded. There is not much justification for subjecting an elderly patient to cataract operation when the vision is normal or practically so in the opposite eye. The patient will not use the eye afterward and will suffer more annoyance than before operation. The attempt to

fit an aphakic eye with an ordinary cataract lens when the other eye sees well is folly. The new type of minimizing lenses offer some hope when their manufacture and fitting become practical.

Light projection and color perception in an eye with mature cataract are important points to ascertain. They give some evidence of the integrity of the retina and optic nerve. It is much better if the surgeon has been able to examine the fun .us during earlier stages when the condition of the vitreous, retina, optic disc and vessels may be noted. In mature cataract the point of fusion and division of two small lights is a little help in determining the soundness of the retina and optic nerve.

Every ophthalmic surgeon makes a preoperative examination of the lacrimal sac, although some limit their efforts to mere pressure over the sac. It is better practice to dilate the punctum slightly and find out if fluid will pass into the nose without pressure. In case the sac is found infected, the safest procedure is to remove it before attempting intraocular surgery. In case of dacryostenosis without apparent infection, probing may be tried. If this fails, ligation of the upper and lower canaliculi by temporary sutures should be done.

The intraocular tension should always be taken with whatever tonometer the surgeon is familiar. In the presence of abnormally high tension one should proceed with infinite caution. One must then decide whether he is dealing with cataract in a glaucomatous eye or with a rise in tension due to an intumescent lens. In either event preliminary iridectomy is indicated if the tension remains high in spite of medical treatment.

The stage of development of a cataract often determines the type of operation that is to be attempted. Certainly in immature cataract intracapsular extraction is the operation of choice. In mature cataract the type of operation depends upon the experience and skill of the surgeon. Many excellent surgeons believe that the capsulotomy operation is safer and yields better results, and this is certainly true in the hands of the occasional operator.

No surgeon has the temerity to open an eyeball in a patient with a dirty, inflamed conjunctiva if it can be avoided. Some still rely upon inspection alone, believing that any apparently clean eye is safe enough to risk operation. Others insist upon blood agar cultures. Conjunctival and epithelial smears which show an absence of leucocytes and pathogenic bacteria have served me well for many years, with the added precaution of the use of 1:5,000 oxycyanide of mercury several days previous to operation.

Knowledge of the state of the blood pressure and urine is essential. Persistent high blood pressure indicates a poor operative risk, and patients of this type should be put to bed for a while under the care of an internist, every effort being made to reduce the pressure before operation. If this is unsuccessful, venesection may be done an hour before operation. It is in this type of patient that the most dreaded of all complications is prone to occur—choroidal or so called expulsive hemorrhage. The use of hypertonic sucrose solution intravenously[1] has been advocated in ocular hypertension, and the reports upon its use are encouraging. I have used it a number of times very successfully. Diabetics of course must be well under control before operation is advisable.

Concerning the elimination of foci of infection before operation, there is a marked difference of opinion. Surgeons of continental Europe pay little or no attention to it, but certainly whenever possible, a foul mouth should be cleaned up. It is not necessary to put an elderly person to the discomfort of having tonsils removed unless they are frankly infected. Undoubtedly an occasional uveitis will follow operation due to their presence, but, on the whole, this seldom happens.

If the bleeding time or clotting time is long delayed, the administration of calcium some days beforehand will often save annoying bleeding at the time of operation

or later. Indeed, the preliminary administration of calcium a few days before operation is a wise precaution in any case.

Within the past fifteen or twenty years several details of technique in cataract operation which have made the operation easier and safer for the average surgeon, have come into general use. Of first value perhaps is the use of hypnotics. By this means the patient may have a good night's rest and come to surgery free from nervousness. It is best to avoid opium in any form unless it has been tried thoroughly on the patient previously, because of the danger of nausea and vomiting. Scopolamine should never be used in elderly persons. Bromides and chloral or one of the numerous barbituric acid preparations are sufficient to allay nervousness and yet maintain the patient's coöperation. Akinesia, either by the Van Lint method of injection of procaine into the orbicularis or the O'Brien method of blocking the facial nerve, is essential, as this eliminates to a great extent the hazard of squeezing. Insertion of a superior rectus suture is a practical measure in patients who have difficulty in controlling the eye movements. Retrobulbar injection of procaine with suprarenin softens the eyeball and lessens the danger of vitreous prolapse. It should be reserved for cases in which intracapsular extraction is to be attempted, as it sometimes softens the globe enough to cause embarrassment in delivering the nucleus in capsulotomy operation. Some form of corneal or conjunctival suture is used by most American ophthalmic surgeons to prevent vitreous and iris prolapse. One or two conjunctival sutures will prevent the folding back of the cornea which occasionally occurs following operation. Corneal sutures may be reserved for unruly patients or those whom it is desirable to allow up very soon after operation.

COMPLICATIONS OCCURRING DURING CATARACT OPERATION

Involuntary Iridectomy. This occurs during the section for a number of reasons. First is probably failure to complete the incision immediately the counter-puncture is made, hence allowing the aqueous to spill early. Pressure with the fixation forceps may force the iris up ahead of the knife. Whatever the reason for the accident, the incision may be completed if an iridectomy was contemplated. Otherwise the knife should be withdrawn and the incision completed with scissors. No harm is done in any event.

Hemorrhage into the Anterior Chamber. This frequently happens and unless it is severe, it need cause no concern. It should be irrigated out before the blood clots, else it obscures the field and makes the manipulation more difficult. The free use of adrenalin before operation will largely prevent hemorrhage from the conjunctival vessels.

Vitreous Prolapse. This is one of the commonest and yet one of the most dreaded of all accidents during cataract extraction. It is especially likely to occur in high myopes and in persons with a previous choroiditis, because in these persons the vitreous is more fluid. It undoubtedly occurs more frequently in intracapsular operations in the hands of the average operator. The posterior capsule, while very thin, exerts quite a restraining influence upon the vitreous body. Heavy handed surgery, especially too much pressure with the fixation forceps is a potent cause of prolapse.

If vitreous presents immediately after the incision and before the lens has been delivered, the surgeon has one of two methods open. He must see that there is no pressure whatever on the globe. An intracapsular capsule forceps may be introduced into the wound, the capsule grasped, and an attempt made to deliver the lens by gentle traction. If this fails, the other recourse is to introduce a wire loop, hugging the posterior capsule of the lens, then lifting the lens forward against the cornea and delivering it. The sutures are then tied and the eye closed. Attempts to remove cortical material should not be made after vitreous has been lost.

It is astonishing what good vision may be obtained after the loss of a comparatively large amount of vitreous. Nevertheless it is a complication that is to be avoided if at all possible. Unwise and persistent attempts to remove a lens in its capsule rather than to do a capsulotomy when difficulties are encountered, accounts for a high percentage of vitreous loss in many series. The idea that it is better to lose a small amount of vitreous to remove the lens in the capsule than to leave some cortex behind is to be condemned. In case the vitreous prolapses after the lens has been extracted, the sutures should be tied, the vitreous cut away with the scissors, and the pillars replaced. During these maneuvers the lids must be held well forward by the assistant to prevent the slightest pressure upon the eyeball, and to produce negative pressure within the orbit. The eye is then gently closed and not examined for four or five days unless the patient complains of pain.

Dislocation of the Lens into the Vitreous. This complication rarely happens at the hands of an experienced surgeon. Not infrequently the zonule ruptures due to too much pressure with the capsule forceps, and the nucleus is dislocated upward behind the iris and ciliary body. It may be easily stroked back into position by gentle manipulation through the cornea above and then delivered in the usual manner. Dislocation deep into the vitreous is a serious complication, and this is more likely to occur in hypermature cataracts with a small nucleus. Removal with a wire loop is the only solution, and this is a difficult feat because the nucleus cannot be seen easily by the operator. Hildreth's ultraviolet light, which produces fluorescence of the lens, is very valuable when this complication occurs.

Expulsive Hemorrhage. This is the most dreaded of all accidents that occur to the ophthalmic surgeon. It is estimated that it happens about once in a thousand operations. This unwelcome complication is prone to occur in patients with both vascular and intraocular hypertension, and careful preoperative preparation will lessen the probability. The hemorrhage comes from the friable choroidal vessels following the sudden release of intraocular pressure. Immediately following the incision the lens may be expelled spontaneously, followed by the contents of the eyeball and a gush of blood. Enucleation is usually necessary following this disaster. Occasionally it occurs a few hours after the operation. The patient then complains of sudden severe pain, often of nausea and the dressing is found soaked with blood. The eye is usually lost. Vail[2] recommends immediate posterior sclerotomy when this accident occurs.

COMPLICATIONS FOLLOWING THE OPERATION

Wound Infection. This usually occurs in patients who have been operated upon in the face of a contaminated conjunctiva. It is manifestly impossible to sterilize the conjunctiva, for saprophytic organisms are always present, and the lid margins and meibomian glands harbor pathogenic bacteria. Nevertheless careful preparation to exclude contamination is usually successful. Improper sterilization of instruments and sutures is a not infrequent cause of wound infection. It occasionally happens that a patient forces infection through a patulous lacrimal duct into an eye by blowing the nose vigorously soon after operation.

In the presence of infection, the patient complains of an undue amount of pain within twenty-four hours. When the dressing is removed, the lids will be found swollen, there is marked chemosis and a slough covered with mucopurulent secretion is found along the upper lip of the incision. Hypopyon occurs early.

Prompt and drastic treatment alone will save such an eye. The wound must be cauterized by trichloracetic acid or the actual cautery, a large dose of typhoid vaccine given intravenously and cold applications used constantly. Sulfanilimide offers some hope in this unfortunate situa-

tion. Usually if the infection is a virulent one, in spite of all treatment, the eye is lost.

Late Infection. This is seen in patients who do not do well after operation. The eye stays more congested than it should. There is pain in or about the eye, and the slit lamp shows all the signs of a more or less severe uveitis. Such patients will usually be found to harbor a focus of infection, and the most common sites in my experience have been in the sequence of teeth, tonsils and sinuses. Treatment with heat, atropine, calcium gluconate and foreign protein will clear up a certain number of them. Removal of infected areas is usually necessary. I have seen two severe, intractable cases respond quickly to sulfanilimide. Some of these patients may be sensitive to lens protein which has been left within the eye. Trauma to the ciliary body in attempting to rupture a zonule which is unyielding doubtless causes uveitis in a considerable number of patients.

Late Hemorrhage. This usually occurs upon the fourth or fifth postoperative day. The patient has been doing well and suddenly complains of a sharp pain in the eye. The surgeon then knows before he opens the eye what he will find. There may be a small amount of blood in the anterior chamber, or it may be filled. While this delays convalescence, it need not cause alarm because it always disappears after a time. This blood comes from a limbal or conjunctival vessel, slipping down through the wound into the anterior chamber. The use of calcium before or just after operation may help prevent this hyphemia. Heat helps hasten its absorption.

Separation of the Wound. This usually occurs during the first few days after operation as a result of retching, coughing or some other strain or by an accidental blow. It is generally accompanied by prolapse of the iris. The patient should be prepared at once for operation as carefully as for the previous one. One or two sutures are placed in the tough conjunctiva directly at the limbus, the prolapsed iris is excised, and the sutures are then passed through the episclera and the wound firmly approximated. A conjunctival flap should then be drawn over the wound. If a few weeks have elapsed and the wound has failed to heal, the edges should be freshened by touching with trichloracetic acid.

Postoperative Psychosis. Elderly persons, placed in unfamiliar surroundings, harassed by the thought of operation upon their eyes, plagued by the fear of permanent loss of vision, and placed in bed with both eyes bandaged, are likely to become confused and even irrational. Much may be done to prevent this by instilling confidence in the patient prior to operation. A nurse well trained in caring for these patients is a valuable asset during the first few days following operation. The unoperated eye should be unbandaged within twenty-four hours following the operation. Sedatives may be used freely, although it must be admitted that some patients do not bear them well and they are sometimes responsible for mental confusion. Very few patients, if properly prepared and given careful postoperative treatment, will become violent. However, should this occur, they must be carefully guarded to prevent injury and if necessary must be restrained. Fluids and sugars should be given freely, and if the patient refuses them by mouth, they should be given intravenously or by rectum. With these precautions the mental equilibrium is reëstablished within a short time.

Eversion of the Cornea. No complication that we encounter gives the surgeon a more unpleasant surprise than to open the eye for the first dressing to find the cornea folded back upon itself with the iris in contact with the upper lid. This cannot happen, of course, if conjunctival or corneal sutures have been used.

W. I. Lillie (quoted by Bothman[3]) has devised an operation by which this may be corrected with excellent results. A conjunctival flap is dissected up from the upper limbus and the original corneal incision is lengthened 1 or 1.5 mm. below the horizontal fold in the cornea. The whole incision is freed with a spatula, prolapsed

iris is excised, and the flap is pulled down over the upper two-thirds of the cornea and anchored at four and eight o'clock. This prevents the high degree of astigmatism that results from replacing the cornea without lengthening the original incision.

Postoperative Glaucoma. Many patients who develop glaucoma following cataract extraction have had it before operation, though it has often been undiscovered. Downgrowth of corneal epithelium into the wound, forming an epithelial lining of the anterior chamber accounts for a few cases. This may be prevented by making a conjunctival flap rather than by ending the incision at the upper limbus. This complication is difficult to diagnose and usually the cause is established by histologic examination. Blocking of the angle of the anterior chamber from a large amount of cortical debris or from the exudate of uveitis may produce secondary glaucoma. An acute rise in tension frequently follows too free discission of soft cataracts. If this happens, it is best to remove the cortical material by linear extraction.

It has been generally taught that glaucoma often is produced by incarceration of a pillar of the iris in the wound. This has been greatly exaggerated as the cause of glaucoma, and such teaching has prevented many ophthalmic surgeons from accepting iris inclusion operations in primary glaucoma. In the light of experience, it seems probable that pillar incarceration following cataract operation in a glaucomatous eye is a safe therapeutic measure.

GLAUCOMA

There are some complications of glaucoma surgery that I do not feel capable of discussing since I no longer do the types of operation in which they occur. A good many years ago I abandoned the trephine operation for the reason that the prolonged absence of anterior chamber formation and occasionally a subsequent opacification of the lens did not appeal to me as an ideal result. In recent years I have confined my operative procedure to four operations, viz.: (1) broad iridectomy if forced to operate during an acute attack; (2) basal iridectomy; (3) cyclodialysis; and (4) iris inclusion. For compensated glaucoma (glaucoma simplex) and as an interval operation in acute glaucoma, iridencleisis has served me well for many years. If done as I shall describe briefly, it constitutes almost a fool-proof operation and there should be no complications.

A retrobulbar injection of procaine and adrenalin is given a few minutes ahead, both for anesthesia and to soften the globe. In case the intraocular tension is unduly high, 50 c.c. of 50 per cent sucrose solution is given intravenously an hour before operation. A generous flap is dissected to the limbus as in the trephine operation. Then a scratch incision about 6 mm. long is made just behind the limbus in the same manner as Elschnig's" iridectomia ab externo." After the aqueous is evacuated, provided miotics have been discontinued for a while and the iris is not too atrophic or bound by peripheral synechia, the iris will prolapse into the wound when the upper lip is depressed. The iris is now withdrawn to the pigmented border and split vertically. One pillar is retained beneath the conjunctiva and the other returns to the anterior chamber. In case the episcleral vessels bleed freely, a pledget of cotton is soaked in adrenalin and allowed to remain beneath the flap a short time before scratching through the sclera. This operation has two features to recommend it over that of the usual operation by keratome incision: (1) it eliminates the danger of scratching the anterior lens capsule with the lance point—in an unusually shallow anterior chamber this may readily occur even in skilful hands; (2) this incision reaches the iris at its base instead of well below it, as occurs when the customary incision is made.

The only complications I have ever encountered in this operation have been: (1) retinal hemorrhages—these are due to the comparatively sudden lowering of

tension in an eye with extremely sclerotic vessels, and may occur during any intraocular operation; (2) hyphemia, which often occurs at the time of operation and disappears within a few days; (3) hemorrhage into the vitreous, which occurs if the incision is made too far back so that after the anterior chamber is evacuated, a bead of vitreous presents, and if the patient is a free bleeder, blood seeps into the anterior vitreous; (4) iritis, probably traumatic in origin and to be combated with atropine and heat. Iritis usually subsides quickly. It is safe to use atropine after iris inclusion operations. Following this operation the anterior chamber reforms within twenty-four hours, massage is started early and continued for some weeks and the results are excellent. I have never seen sympathetic ophthalmia nor late infection develop after this operation, and I believe it is the safest and best filtering procedure yet devised.

This by no means covers all the complications that the ophthalmic surgeon falls heir to as a part of the hazards of his chosen profession. It has been my purpose to point out ways to avoid complications rather than to stress treatment after they occur. The best way to get out of trouble is not to get into it. An old sage once remarked that "the man who makes no mistakes does no business." Just as surely, the man who has no complications does little surgery. Some of them are unavoidable; most are preventable. The surgeon who selects his cases wisely, prepares them properly, uses the technique that is safest in his hands and supervises the postoperative care with minute attention to detail, will escape much of the trouble that arises to plague his more careless colleagues.

REFERENCES

1. Dyar and Matthew. *Arch. Opbth.*, 18: 57, 1937.
2. Vail, D. *Am. J. Opbth.*, 21: 256, 1938.
3. Bothman, L. *Arch. Opbth.*, 17: 1073, 1937.

THE CONJUNCTIVAL FLAP IN OPHTHALMIC SURGERY

JOHN GREEN, M.D.

Professor of Ophthalmology, St. Louis University School of Medicine

ST. LOUIS, MISSOURI

A CONJUNCTIVAL flap is most frequently used as a part of the modern technique in cataract surgery. It has long been recognized that a flap of generous dimensions promotes prompt agglutination of the lips of the wound and thus provides a safeguard against exogenous infection. It is also realized that a sutured flap holds the lips of the wound in closer apposition during the early postoperative period and thus tends to lessen the danger of prolapse of the iris.

A detailed description of the various flaps proposed for this purpose would be tedious and outside the scope of this paper. Suffice it to say that all could be placed in one of the following classes.

1. A flap formed by the Graefe knife at the completion of the corneal section. This should be reasonably large and should include conjunctiva at both ends of the section. Some surgeons are content to leave the flap unsutured, but it is the usual practice to bring the edges of the flap together with accurately placed interrupted sutures or with a continuous suture.

2. A flap is formed by scissors dissection prior to the section. This has the advantage of insuring the size and shape of the flap. Usually sutures (from two to five) are inserted (but not tied) prior to the section.

3. The "pocket" flap (proposed by Spratt) is formed by a horizontal incision through the conjunctiva 7 or 8 mm. above the superior corneal limbus. By dissection under the flap access to the lateral aspect of the limbus permits the usual section with the cataract knife. At the completion of the operation the flap is tied with a single mattress suture.

4. The "apron" flap (Van Lint). The conjunctiva is incised close to the upper corneal margin, the incision comprising about three eighths of the corneal circumference. The flap is freely undermined up to the upper fornix. Lateral sutures are inserted near the ends of the flap. At the end of the operation the flap is drawn down over the incision and held in position by inserting needles through the conjunctiva and episclera at 4 and 8 o'clock respectively.

5. The corneo-scleral-conjunctival flap. To secure more positive apposition of the lips of the section, Verhoeff uses two sutures which pass through corneal and scleral tissue. After emergence the sutures pass through the conjunctiva which has previously been dissected back from the upper limbus. When tied, the corneal section is securely sealed and the conjunctiva is drawn down to cover the section.

Rupture of the wound after extraction of the lens is a formidable accident. It may result from sneezing or coughing, a blow by the patient himself, straining, squeezing the lids, or accidental pressure on the globe. When such an accident occurs the corneal lip is elevated, being pushed forward by the prolapse of a portion of the iris, the vitreous, or a hematoma. A horizontal groove crosses the cornea. Unless the eye is subjected to further operative interference, a high degree of astigmatism will result and there may persist a linear opacity corresponding to the groove in the cornea which further interferes with vision.

Bothman[1] has modified Elschnig's[2] procedure as follows: A conjunctival incision is made at the limbus from 4 to 8 o'clock. The conjunctiva is freely dissected from the upper half of the globe. Sutures are placed opposite 10 and 2 o'clock and fixed in the bulbar conjunctiva about 4 or

5 mm. below 4 and 8 o'clock. The lips of the wound are separated at both lower angles. The corneal section is then enlarged for from 1 to 1.5 mm. below the horizontal groove. The prolapsed iris, if it cannot be easily replaced, should be excised. If a hematoma is present it should be removed. The remainder of the incision is opened with an iris spatula and the cornea stroked into proper position and held there while the conjunctival flap is pulled down from above and sutures tied. With this method Bothman claims that healing ensues with a minimum of postoperative astigmatism. Von Herrenschwand[3] is of the opinion that this complication can usually be forestalled by covering the wound at the time of operation with a prepared flap of conjunctiva.

Always a difficult problem is extraction of cataract in an eye that has a filtering bleb following a trephine operation, especially if the trephine opening is at the superior corneal limbus. Elliott[4] found that a section which included the conjunctiva over the trephine opening was apt to heal with closure of the drainage canal. Nor was he satisfied with a corneal section which is attended with a risk of delay in the healing. He has tried and found satisfactory the following procedure: the cornea is transfixed in the usual way with a cataract knife. The section is continued until nearly clear of the filtering pad. The knife is then turned to make a broad conjunctival bridge, either upward and outward or upward and inward to the outer or inner side of the filtering scar.

A very useful operation in which a conjunctival flap is the principal feature is that proposed by Wheeler for the treatment of hypotony following filtering operations. In this operation a crescent of cornea at the upper limbus is removed with a curette. The flap covering the trephine hole is dissected up and sutures are inserted laterally to draw the apron down over the cornea. The flap adheres to the scarified cornea and normal tension is restored.

Federici,[5] on the basis of experimental work on the histology of the conjunctival plastic operation, draws the following conclusions: (1) The conjunctival flap aids actively the process of repair of large perforating wounds of the cornea, but it is necessary that the wounds be of very recent date. (2) The attachment of the flap is more rapid and the healing of the wound occurs with a more solid cicatrix when the flap is furnished with abundant conjunctival tissue. (3) In linear wounds in general and extensive wounds of the cornea several days old, the conjunctival flap usually does not take part in the process of repair, and when freed of sutures, it retracts completely. (4) Failure of attachment of the flap is due to the fact that the epithelium of the cornea, rapidly regenerating, excludes the conjunctiva from all contact with the lips of the corneal wound. (5) There is in every case attachment of the transplanted conjunctiva, when, between the edges of the corneal wound, there is fresh iris tissue. This tissue separates the edges of the wound and adheres readily to the conjunctival tissue, thus hindering the rapid epithelialization of the wound itself. (6) When the flap attaches, the posterior surface is covered, at first, with an epithelial stratum which tends to join the corneal epithelium emanating from the lips of the wound. Later, the flap proliferates actively by replacement of the fibrinous coagulum which carpets the corneal wound with new formed connective tissue. The flaps of the cornea show only a slight tendency to neoformation. When closure of the wound has thus occurred the flap undergoes rapid atrophy so that eventually it is barely perceptible. (7) In cases in which the flap does not attach, it has no direct action on the process of repair. (8) The conjunctival plastic operation, even when the conjunctiva does not participate directly in the process of repair of the corneal wound, influences the course of the wound favorably, being especially useful in cases in which there is danger of infection.

L. de Souza Queiroz[6] discusses various types of conjunctival flaps and illustrates them by case reports. He advocates total coverage by circumcision of the cornea and the use of a purse-string suture. Partial covering by sliding is especially indicated in marginal injuries or ulcers of the cornea. The conjunctiva is incised next to the margin and is dissected back as far as possible. It is then slid over the cornea and sutured with two stitches at the extremities of the incision. When the conjunctiva is friable and when a permanent covering is desired, Queiroz offers an ingenious modification. The conjunctiva is incised along the limbus for about half the circumference. At one of the extremities of the incision another is made, vertical to the limbus, about 5 mm. long and at the extremity of this a third incision is made parallel to the first. A flap is thus obtained which should be dissected as far as possible. A tunnel is then prepared under the conjunctiva along the second incision. A mattress suture carries this flap under the conjunctiva in a manner similar to McReynold's operation for pterygium. When the suture is tied the conjunctiva slides over the cornea, covering the lesion and creating adhesions. Similar suggestions were recently made by W. Wallace Hoare[7] and Szokolik.[8]

Sometimes when this buried flap is used it is found that the conjunctiva is stretched too much, thus tending to rupture the sutures and retract the flap. In this case an incision is made with scissors parallel to the first, transforming the flap into a true bridge across the cornea. The bridge flap is indicated principally in cases where the corneal lesion is not extensive and is centrally or paracentrally situated. Some time later, when the cicatrix has formed, the band may be resected from both sides, leaving only a small portion of the conjunctiva adherent to the cornea. The flap should be made in the vertical direction so that movement of the lids will not tend to dislocate it.

Schmidt[9] discusses the technique of Kuhnt's use of conjunctiva. The suggestions made apply chiefly to what is called "conjunctival covering" in which the conjunctiva is used only for temporary closure of the wound. The first requisite is that such a flap should contain no subconjunctival tissue which interferes with the elasticity of the flap so that the edges cannot be brought together without traction. To secure this very thin flap, Schmidt has devised a special forceps that picks up only the superficial layers of the conjunctiva. He dispenses with scissors and cuts the conjunctiva with a special knife which is two-edged and tongue shaped, 2 mm. wide at the base and tapering to 1 mm. at the rounded but very sharp tip.

Wright[10] states that, according to the nature of the condition, a conjunctival flap acts either temporarily as a protective covering or permanently as a graft. Care should be taken not to use such a flap in vascular conditions of the cornea lest it form undesired adhesions. An apron flap cut from near the limbus is usually intended to act as a graft, becoming permanently attached and forming a sound protective covering for the defect.

Wright describes an "unorthodox" method of treating a corneal fistula. The epithelium of the opening is destroyed by the application of a fine electrocautery point. A pedicle ribbon flap is raised from the upper bulbar conjunctiva. This is carried down across the defect and stretched out thin and level over the fistula, its free end being buried in a subconjunctival pocket at a point diametrically opposite its attached end. A fairly flat contact glass is applied and not removed for seven days. On removing the glass, Wright found that the flap had adhered. The unadherent portion was carefully cut away.

In another case a thin epithelial ribbon was taken from the inner side of the arm. This was laid over the prepared floor of the ulcer and the ends of the ribbon

buried beneath the conjunctiva at either end of the corneal diameter. Wright emphasizes an additional point, viz., that whenever the ulcer is close to the limbus a narrow track should be scratched from the ulcer to the limbus. Such a track is outside the visual zone and gives the graft a much better chance for vascularization. Wright even advocates this "rawing" of the cornea when the defect is near the center.

In wounds of the sclera there is always the problem whether the sclera should be sutured or conjunctiva alone should be used for approximation and coverage. However, it is possible to combine the two procedures. In small scleral wounds the double overlying conjunctival flap first proposed by L. M. Francis is probably the best method. The conjunctiva on either side of the scleral wound is dissected up and a mattress suture placed in each lip. One of the flaps is buried under the other. If the proper amount of traction is used the scleral wound coapts very perfectly. Large jagged wounds of the sclera are best approximated with fine catgut sutures and covered with conjunctiva, care being taken that the suture line of the conjunctiva does not coincide with the scleral wound. In extensive wounds of the cornea, especially where the corneal flaps are jagged and irregular, it is usually best to cover the entire cornea. This, in my experience, is best done by circumcising the cornea, freely undermining the conjunctiva and inserting a purse-string suture. When the suture is tightened the entire corneal area, save for a minute portion in the center, is completely covered. Unless infection ensues, the corneal flaps hold surprisingly well. In such extensive wounds there is usually an associated laceration of the iris and injury to the lens so that the visual outcome is not satisfactory.

Within the past ten years there has been a revival of interest in keratoplasty and several new techniques have been developed. The necessity of anchoring the transplanted disk in the operation of partial penetrating keratoplasty has been met by the use of conjunctival flaps. Filatow[11] makes in the upper segment of the globe a conjunctival flap 6 mm. wide and 10 to 12 mm. long. At the lower edge of the cornea an incision 6 mm. wide is made in the scleral conjunctiva. After the transplant has been placed in the aperture the conjunctival flap is laid upon the graft with its epithelial side down and it is sutured into a conjunctival incision at the lower edge of the cornea. The flap is left in position for two or three days.

Castroviejo[12] makes conjunctival incisions above and below near the limbus, leaving there two zones, one on each side of the horizontal meridian through which the conjunctival incisions do not reach. After the implant has been made the conjunctival flaps are pulled over the graft and sutures, four in number, are tied.

Nizetic[13] has concerned himself with the fixation of the graft in keratoplastic operations. In his opinion the best type of fixation is with a reversed flap of conjunctiva, the epithelial side being placed next to the cornea. The flap is 6 to 10 mm. wide and 10 to 11 mm. in height. It is removed from the upper limbus in an upward direction, is turned, and is firmly attached at the lower limbus to a freshened conjunctival wound in such a way as to be stretched over the graft. He makes the point that the epithelial side of the flap does not irritate the implant. As early as 1877 Schöller first proposed this type of flap; in fact, he was the first to use conjunctival flaps extensively. Nizetic emphasizes the necessity of preparing a flap with a subconjunctival tissue attached so that it may resist the tearing out of the sutures.

Stallard[14] states that Rycroft uses a complete conjunctival flap drawn over the cornea and implant and retained in place with a purse-string suture. Stallard approves of this method and uses it in his own work. It is supposed that during the five or six days in which the

flap is in place it pours out serum and lymph, possibly affording nutrition to the graft. Stallard also quotes Somerville-Large as approving of a large conjunctival flap drawn from above downward to cover the graft and held in position by two mattress sutures.

B. Rosengrin[15] describes two types of conjunctival flap which he uses in corneal transplants. The first is a simple double pedunculated flap and the second is a flap with a single pedicle which is sutured to the conjunctiva on the opposite side of the cornea.

In 1936 Francois[16] advocated for the treatment of marginal degeneration of the cornea the excision of the ectatic portion and covering the wound with a conjunctival flap.

Rubbrecht[17] states that he has been dissatisfied with the treatment of progressive corneal ulcers. The problem is to arrest and cicatrize a progressive ulcer before it has invaded the pupillary zone and to cure it with the least amount of leukoma possible if the center of the cornea is already involved. Rubbrecht's method is based on the well-known fact that the progress of an ulcer is arrested after spontaneous perforation or when new formed vessels come from the limbus in sufficient numbers. The procedure is as follows: The ulcer is curetted and the necrotic tissue removed. The ulcer is incised, thus permitting better apposition of the edges of the incision which goes through the entire thickness of the cornea and extends to sound tissue on either side. If at all possible, the center of the cornea should be avoided. The conjunctival flap is in the form of a bridge which is dissected up and laid over the cornea, thus covering the ulcer completely. It is held in position by sutures appropriately placed. The bridge is not cut until two or three months after the operation. Rubbrecht believes that all progressive ulcers of whatever nature should be treated by this method.

Kreiker[18] points out that one of the contributing factors in rodent ulcer is the relatively unprotected state of the cornea. For this reason he has advocated and practiced conjunctivoplasty. Merely covering the ulcer is not enough. If the unaffected portion of the cornea remains uncovered there will soon be a groove-shaped recurrence at the edge of the adherent flap, probably because the conjunctiva never heals in an exact line around the edge of the ulcer, but shrinks somewhat and so leaves a small diseased margin exposed. Under complete coverage, there is immediate and certain cessation of ulcer formation. Cases are cited to show the failure of partial coverage and the success of complete coverage. Kreiker does not use the purse-string, but draws down a very large flap which is detached high up and laterally. After the ulcer is scraped with a curette, the conjunctiva is drawn downward and sutured to the conjunctiva and episclera at 6 o'clock. Two or three additional sutures are placed lateral to this one. The author believes that one need have no misgivings about the fate of the remaining intact epithelium or the cornea which lies under the conjunctival flap, as the flap adheres only to the territory of the ulcer. Eventually the conjunctiva should be slit and allowed to retract.

Peterson[19] has had considerable experience with iris prolapse following perforating ulcer. The lesion was located in the upper half of the cornea in thirty-six out of thirty-eight cases. He did not favor the pedunculated flaps described by Kuhnt which were discarded in favor of a flap formed by undermining a large apron of conjunctiva and drawing it down over the corneal perforation, after excision of the prolapse. The flap, sutured with two mattress sutures should reach 3 mm. below the corneal defect without undue tension. He points out there must be neither too superficial dissection of the flap nor too deep, but that it certainly should contain some subconjunctival tissue. The immediate results from the procedure were very encouraging. Of thirty-eight cases of hernia

of the iris, twenty-nine had a primary attachment at the first operation. Of nine cases in which a second operation was advised, six consented and five had a successful result.

Majewski[20] contends that there is no better basis for a prothesis than the patient's own blind eye, provided it be covered by conjunctiva. The eye, however, must fulfill certain conditions. First, it must be smaller than the other eye so that there is enough space for the prothesis. Second, it must not be sensitive to touch. Third, it must be deprived of its epithelium and covered with conjunctiva. The operation is as follows: The corneal epithelium is rigorously curetted, followed by scarification with a small pointed knife. The dissected flap of conjunctiva is then drawn over the cornea so that the upper flap covers the lower, the flaps being fastened with sutures above and below. The artificial eye can be fitted in three weeks from the time of operation.

Tjanides[21] has used conjunctival flaps in all serious corneal injuries, whether or not infected. Uniformly satisfactory results led him to consider using the method in severe infections of the conjunctiva threatening the cornea. It seems startling to think of surgery in the presence of such an infection as gonorrheal ophthalmia. Nevertheless, he was able to establish the fact that a flap had a definite and important rôle in protecting the cornea in this infection. He was able to protect the cornea by total conjunctival covering executed during full suppuration from gonorrheal conjunctivitis and in a completely septic field. He contends that the time to perform the operation is when the cornea first begins to cloud. The few days during which the cornea is completely covered gives time for a vigorous attack on the gonococcus.

Recently, he has supplemented the simple procedure by interposing between the cornea and the flap a fragment of amniotic membrane taken from a healthy mother. His point is that by the use of this membrane the protection of the cornea is more complete. It was observed in the days following the operation that with the progressive loosening of the suture the interposed amniotic membrane prevented the contact of the cornea with the actively secreting conjunctiva. The author suggests that in place of amniotic membrane, a dermo-epidermic graft taken from the thigh might serve the purpose quite as well. Though recognizing the danger of dissemination of the infection by the passage of the gonococci into the circulation through opened vessels, he asserts that the danger of destruction of the cornea and subsequent blindness is so great that any means which prevents corneal involvement is justified. If dissemination should occur, it is unfortunate, but far less so than loss of sight.

In my own experience conjunctivoplasty has proved especially valuable in the following types of cases: marginal ulcer of the cornea, recurrent pannus in chronic trachoma, serpiginous ulcer, hernia of Descemet's membrane, perforation of the cornea following syphilitic keratitis, ulcer of the Mooren type, and ring ulcer of the cornea. In each case I try to individualize the type of flap used. In the majority of my own cases I have used the complete coverage by means of a purse-string suture or various types of sliding flap. I feel that the pedunculated or bridge flap of Kuhnt has a very limited usefulness.

CASE REPORTS

Case 1. Complete coverage of the cornea. Twenty years ago, the patient, a deaf mute, passed through a severe keratitis with iritis. Increased intraocular pressure required an iridectomy. This relieved the pain but the eye gradually became totally blind, possibly from detachment of the retina. Recently she reappeared complaining of a constant tearing and scratchy sensation. Examination with the slit lamp revealed a very uneven cornea with numerous thick calcareous plaques which projected above the surface. The superficial layers of the cornea were carefully dissected off. The conjunctiva was freed from the limbus and a

purse-string suture inserted, the flap covering the entire denuded area.

The result has been satisfactory. There is no longer any unpleasant sensation in the eye. The tearing has stopped entirely.

Case II. Penetrating wound with iris prolapse.

A male, aged 29, was struck in the right eye by the end of a telephone wire which snapped back while he was unrolling it from a drum. There was a perforation at the limbus with small prolapse of the iris. The patient was seen twenty-four hours after the injury. There was no pain and no infection. The iris was grasped with forceps, drawn out, and snipped off. A sliding flap was drawn over the wound and sutured. Healing was uneventful with restoration of normal vision.

Case III. Inveterate pannus and marginal keratitis.

R. W., male, aged 38 years, was first seen December 27, 1923. He had long been a victim of chronic trachoma. Recurrent attacks of pannus accompanied by small ulcers compelled him to stop work for two or three weeks, and as these attacks came several times a year, they seriously interfered with his work, that of a machinist's helper on a railroad. Local treatment, which consisted of holocaine, hyoscine, silver nitrate, and scarlet red salve, failed to eradicate the pannus or prevent ulceration. Pain was a conspicuous feature, and by reason of its frequent recurrence and long continuance was rapidly undermining the patient's morale. The entire area of the pannus and ulcer at the upper limbus was curetted. Partial peridectomy was performed at this site and a conjunctival flap was drawn down to cover the curetted area. A marginal ulcer from 7 to 9 o'clock was curetted and another small conjunctival flap was drawn up to cover it. Recovery was uninterrupted. The patient was seen again two years later. His eye had remained entirely free from ulcers or pannus. There was a small conjunctival cyst at 9 o'clock, adjacent to the cornea. This was removed, and one suture was inserted. V. = $\frac{6}{10}$.

Case IV. Serpiginous ulcer.

E. N. T., male, aged 59 years, had had recurrent attacks of ulceration of the right cornea for twelve years. The present trouble began two months ago. Treatment at an eye clinic for seven weeks was without improvement. The right conjunctiva was inflamed and velvety. An irregular, serpiginous ulcer occupied the central portion of the cornea. The general condition was fair; Wassermann, negative. Local treatment consisted of silver nitrate, 1 per cent, scrubbed on the upper lid; holocaine, 1 per cent; atropine and cocaine in oil three times a day; dry heat; and boric acid flush. The conjunctiva adjacent to the lower corneal margin was incised and the flap drawn upward and held by several interrupted silk sutures. The following day, two of the sutures had pulled out. They were reinserted, and the flap drawn up again. The operation was followed by immediate relief from pain. Four days after the first operation the flap had almost entirely retracted. The staining area had diminished about 50 per cent. Ten days later there was a slight extension of the denuded area, accompanied by pain. This yielded to routine treatment. When the patient was last seen, the eye was white and quiet. V. = $\frac{6}{32}$.

Case V. Ulcer of Mooren type—Spastic entropion—Iritis.

J. M. L., male, aged 58 years, in March 1924 found his left eye inflamed and painful. The patient believed this was caused by some embalming fluid entering the eye. Treatment covering a period of six weeks had not helped. Examination showed a spastic entropion and a marginal ulcer with undermined edges running from 4 to 8 o'clock. The depth of the latter, its situation, and ragged overhanging edges suggested an ulcer of the Mooren type. There was diffuse staining of the lower half of the cornea and a low-grade iritis.

The usual local treatment was unsuccessful. Several badly ulcerated roots were extracted. The patient was given several milk injections with the usual systemic reaction, but without improvement in the ocular condition. A semilunar strip of skin was excised from the lower lid, the entire ulcerated area was curetted, and a conjunctival flap was drawn up to cover the entire site of the ulcer. Recovery was uneventful. The flap retracted partly, but remained adherent over the site of the ulcer. The patient was seen a year later, at which time the eye was entirely well, but vision, owing to an exudate in the pupil, was only $\frac{1}{50}$.

Case VI. Ring ulcer of the cornea.

W. A., aged 30 years, consulted me several years ago on account of bilateral trachoma. At this time the right upper eyelid was much thickened, with the formation of scar tissue

and entropion. There was pannus, with repeated ulceration. In the left eye the conjunctiva was thick and velvety but there was no pannus. Excision of the tarsal cartilage of the right eye was followed by prompt improvement and ultimate cure of the trachoma. The condition of the left eye did not justify operation at that time, and consequently medical treatment only was resorted to. When last seen both eyes were quiet and it appeared that the trachomatous process in the left eye was quiescent.

About August 15, 1925, the left eye became inflamed and painful and the patient thought a cinder had entered the eye. On September 1, 1925, during my absence, he was seen by my assistant, who found an acutely inflamed eye with pannus and ciliary injection. The patient was advised to remain for treatment but declined to do so. He returned to his home town, where a local oculist performed an excision of the tarsal cartilage. Apparently loop sutures with the knots on the conjunctival surface were used, as there was an extreme amount of irritation and pain following the operation, and narcotics had to be resorted to. The sutures were removed at the end of the third day but the patient's suffering continued unabated. Finally, on September 21, he returned to St. Louis. The condition at this time was desperate. The entire periphery of the cornea was occupied by a deep ring ulcer, which had destroyed all but a little island of epithelium not more than 2.5 mm. in diameter in the center of the cornea. Fortunately, perforation had not taken place, and the pupil which could be dimly seen, was round and active. The patient had spent several sleepless nights, and was much prostrated by his suffering.

The ulcer was lightly curetted. The conjunctiva was completely circumcised, freely undermined, and a silk purse-string suture run through the edge of the flap thus formed. When the purse-string was drawn taut, the entire corneal surface was covered by the flap. The patient spent a comfortable, almost painless, night, and made an excellent recovery, but the conjunctiva became adherent to all but the central part of the cornea. It was interesting and surprising to note the increasing thinning and transparency of the membrane, so that it became possible to discern easily the texture of the iris and the motility of the pupil. Needless to say, vision was low, $\frac{1}{60}$.

Later, I dissected off the flap. It was loosely adherent in all but two or three places. Owing to the large amount of irregular astigmatism, much visual benefit is not to be expected, but the improvement in the appearance of the eye is noteworthy.

SUMMARY

A conjunctival flap provides a safeguard against exogenous infection and is almost universally used in the cataract surgery of today.

In rupture of the wound after extraction of the lens, the use of an apron flap of conjunctiva combined with enlargement of the wound is suggested.

A lateralized conjunctival bridge is suggested by Elliott when a cataract operation is necessary in an eye with a filtering bleb.

An apron flap combined with scarification of a crescent of cornea is useful to correct excessive hypotony.

A sliding flap may be sutured to the conjunctiva and episclera. To insure adhesion and prevent premature cutting out of the sutures, the ends of the flap may be buried under a small tunnel of conjunctiva.

The corneal implant in keratoplasty is most frequently stabilized by some type of conjunctival flap.

A bridge flap has been found useful in progressive ulceration of the cornea.

Rodent ulcer may be covered with a flap which must be larger than the size of the lesion.

A shrunken blind eye with intact cornea may sometimes be used as the basis for an artificial eye by covering the denuded cornea with a complete conjunctival flap.

Complete coverage of the cornea has been found to prevent ulceration in acute gonococcal ophthalmia.

REFERENCES

1. Bothman, L. Repair of rupture of the wound after extraction of cataract. Report of cases. *Arch. Ophth.*, 17: 1073–1075 (June) 1937.
2. Elschnig, A. Augenärtzlichen Operationslehre. Graefe-Saemisch Handbuch der gesamten Augenheilk. 1922, vol. II, page 1093.
3. v. Herrenschwand, F. Die Wundsprengung nach alter Star-Extraktion und ihre Verhütung durch

die Anlegung eines vorbereiteten Bindehautlappens. *Ztschr. f. Augenheilk.*, 83: 87–94, 1934.
4. Elliott, R. H. Choice of operation for cataract on a previously trephined eye. *Brit. M. J.*, 1: 958–959, 1933.
5. Federici, E. Su alcune particolare indicazione dell' autoplastica congiuntivali nell' operazione di cataratta. *Arch. d. ottal.*, 39: 417–438 (Sept.-Oct.) 1932.
6. Queiroz, L. de Souza. Recobrimento conjunctival. *Arq. Inst. Penido Burnier*, 3: 197–203. (Dec.) 1934.
7. Hoare, W. Wallace. Letter to *Brit. J. Ophth.*, 19: 235, 1935.
8. Szokolik, E. The technique of conjunctivoplasty. *Am. J. Ophth.*, 11: 438–439 (June) 1928.
9. Schmidt, H. Zur technischen Weiterbildung der Kunstchen Bindehaut Verwertung. *Ztschr. f. Augenheilk.*, 81: 239–243, 1933.
10. Wright, R. E. The contact glass as an aid in corneal plastic surgery. *Brit. J. Ophth.*, 16: 473–475 (Aug.) 1932.
11. Filatow, W. Zur Technik der Parziellen durchgreifenden Keratoplastik. *Ztschr. f. Augenheilk.*, 65: 147, 1928.
12. Castroviejo, R. Keratoplasty; an historical and experimental study including a new method. *Am. J. Ophth.*, 15: 825–838, 905–916, 1932.
13. Nizetic, Zdravko. Fixation des Transplantates bei Keratoplastischen Eingriffen. *Ztschr. f. Augenheilk.*, 90: 305–309, 1936.
14. Stallard, H. B. Corneal grafting (keratoplasty): some modifications in technique. *Brit. M. J.*, 1: 106–109 (Jan. 18) 1936.
15. Rosengrin, B. Bidrag Till Korneal Transplantationens Teknik. *Hygeia*, 92: 826–828, 1930.
16. François, J. Marginal degeneration of the cornea. *Arch. d' Ophtalm.*, 53: 432, 1936.
17. Rubbrecht, M. R. Un nouveau traitement des ulcères progressifs de la cornée. *Bull. et. mém. Soc. franç. d' opht.* 44: 331–344, 1931.
18. Kreiker, A. Vollstaendige Bindehautdeckung der Hornhaut als Heilmittel des Ulcus Corneae Rodens. *Klin. Monatsbl. f. Augenheilk.*, 90: 373–377, 1933.
19. Peterson, R. A. Iris prolapse from corneal ulcer; treatment by conjunctival flap. *Am. J. Ophth.*, 11: 979–981 (Dec.).
20. Majewski, K. W. Syndesmoplastie epicornéène à l' usage de l' epiprothèse. *Arch. d' Opht.*, 45: 673–680 (Nov.) 1928.
21. Tjanides, T. De la protection de la cornée dans l' ophtalmie gonococcique. *Bull. Soc. d' opht. de Paris*, pp. 466–472 (Oct.) 1930.

Rokitansky (1804–1878, Vienna) developed the first complete and systematic method of postmortem examination, and personally performed more than thirty thousand autopsies.
From—"The Postmortem Examination" by Sidney Farber (Thomas).

ECTROPION AND ENTROPION OF THE EYELIDS

WEBB W. WEEKS, M.D.

Professor of Ophthalmology, New York University School of Medicine

NEW YORK CITY

ECTROPION

WHEN an eyelid has acquired or assumed during its development a position which interferes with its functional activities, or if such a condition has resulted from disease processes or injury, especially if pathologic changes have set in, surgical measures are indicated.

A spastic rolling outward of the eyelids occurs through irritative inflammatory changes in the eyelid structures or parts adjacent. Unless relieved, structural changes occur in the skin, muscular, fibrous (tarsal) elements, and conjunctiva, which give a permanency to the deformity and cause the loss of function of the eyelid.

Preceding the surgical procedures advised, aseptic preparation of the parts involved have been carried out and local anaesthesia instituted. For the conjunctiva local instillations of pontocaine 1 per cent is used and an infiltration of the eyelid is made with 2 per cent novocaine with adrenalin added.

Should the ectropion be more or less recent, yet failing to be relieved by local non-surgical measures, cautery punctures should be tried. The millimeter tip of an electric cautery needle at a dull red glow is carried through the conjunctiva and into the tarsus 3 mm. from the sharp posterior margin of the eyelid, spaced 6 mm. apart along the length of the tarsus temporal to the punctum lacrimalia. A second row 3 mm. further posteriorly may be made.

Another procedure of value is placing two double armed No. 3 twisted black silk sutures, starting high on the exposed conjunctival curve: one at the junction of the middle and inner thirds; the other at the junction of the outer and middle thirds of the eyelid. The needles are carried down to the tarsus, along whose posterior surface they travel to its inferior border, then forward through the eyelid structures to the orbit margin. Here the needles pick up the periosteum and continue on through the skin. The sutures are then tied over small gauze rolls firmly enough to invert the rolled out portion of the eyelid.

Should the conjunctiva be markedly thickened, an elliptical 4 mm. portion, 3 mm. posterior to the posterior sharp margin of the eyelid and extending the length of the tarsus, may be excised. The cut edges are approximated by interrupted black silk sutures.

A relaxed, stretched out lid occurring in an old ectropion with atonic and atrophic structural changes requires shortening and drawing up the eyelid so that the sharp posterior lip hugs the eyeball. Such conditions are seen in senile atonic states and eyelid conditions following facial nerve paralysis. The Kuhnt-Szymanowski operation will correct this condition:

First Stage. Intermarginal incision, splitting of the lid, either with a U-shaped lid clamp or between the fingers, beginning at the junction of the middle and inner thirds of the margin and running to the external canthal angle, through the depth of the lid.

Second Stage. Two forceps, grasping the tarsus in the middle area, fold it to determine the width necessary to remove to bring about the desired correction. This section, 5 or more mm. in a v shape including only tarsus and conjunctiva, is cut out.

Third Stage. An incision through skin starting at the external angle is then cut 5 mm. or more obliquely up and outward; another incision twice this length is cut

from the external angle down and slightly perpendicular to line of first incision. The extremities of these two incisions are united

Fig. 1. Senile ectropion.

by an incision and the included skin flap excised.

Fourth Stage. Five mm. or more of the outer margin of the lower lid is denuded of its cilia and ciliary follicles. An assistant grasps the lid margin in the middle and partly everts it. Silk sutures are then placed in the tarsus from below up, running from without inward; they are three in number, the top one catching tarsal edges quite near the upper margin. These sutures are tied, allowing the lid to stand upward. A suture now joins the apex of the lower lid flap with the upper end of the triangle of excised skin. Subsequent sutures unite the skin edges where necessary. If the parts have been removed equally, the lid stands up in position, drawn up against the eyeball and outward. It may be necessary to close a slight gaping of the margin wound, which can be done by a suture placed just below the cilia, 4 mm. away from the tarsal wound and run from without it, coming back out 3 mm. away and tying over a pig. A firm dressing is applied.

For a partial ectropion of some standing the procedure of Imbré is advised, as follows:

First Stage. Split the lid in the gray line in the area of most prominent sag of the lid, depending on the amount and position of the variant from the normal position—10 to 16 mm.

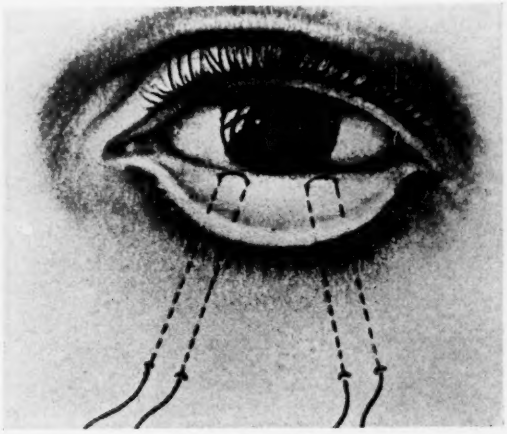


Fig. 2. Placement of Snellen sutures.

Second Stage. At the nasal end of this split cut out a triangle of tarsus and conjunctiva with the base 3 to 6 mm. on the

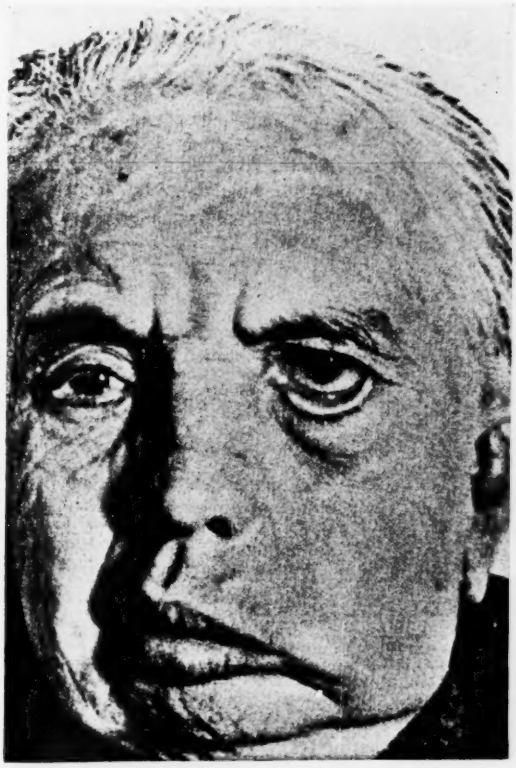
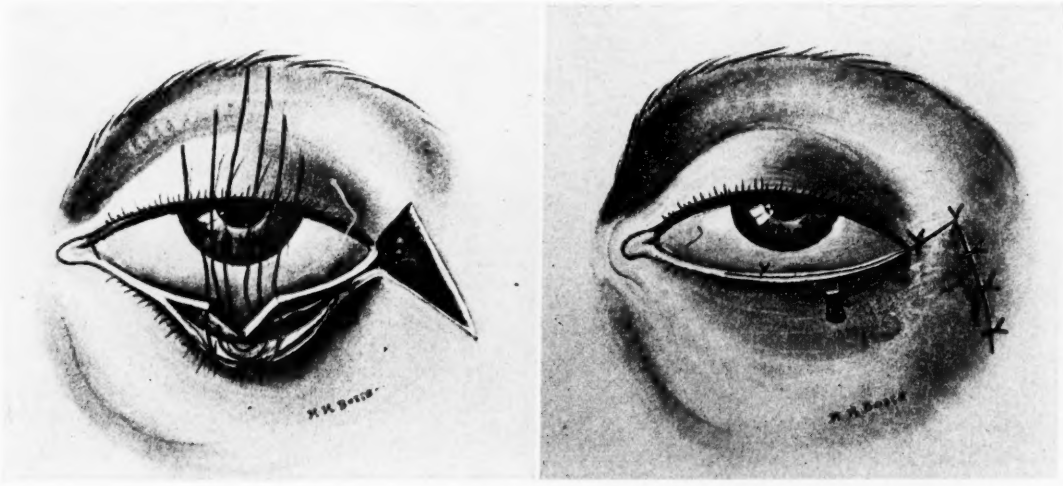


Fig. 3. Paralytic ectropion.

lid margin and an apex 5 mm. toward the cul-de-sac.

Third Stage. At the temporal end of the split lid margin make a vertical cut

through the ciliated portion 3 mm. long. From this vertical cut running nasally make a cut through the skin and orbicularis muscle parallel to the lid margin 10 to 16 mm. long. A triangle of skin and muscle, with 3 to 6 mm. at the base is cut, with the apex downward 5 mm.



A B

FIG. 4. Kuhnt-Szymanowski operation, fourth stage. A, tarsal sutures. B, suturing of skin flap.

FIG. 5. Cicatricial ectropion.

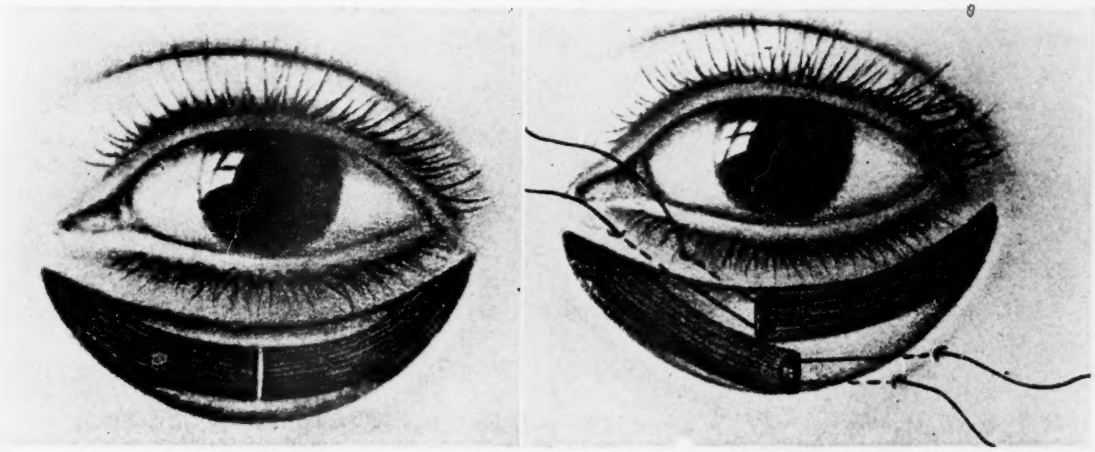
Fourth Stage. The tarsal triangle is closed with three interrupted silk sutures tied on the conjunctival side. The top suture is close enough to the margin to approximate it accurately.

Fifth Stage. The skin and muscle triangle is then closed with three interrupted sutures. This causes a sliding of the 3 mm. ciliated skin and muscle marginal strip so as to overlap the temporal end of the lid split. This overlap is excised and the flap edges closed by interrupted silk sutures.

Should the ectropion follow a cicatricial contraction due to a second degree burn, injury, or structural changes occurring in eczematous or lupoid disease of the skin, the loss of skin has to be made up by skin grafting. The skin of the upper lid of the same or opposite side, when available, is the best for this purpose. When not available, a Thiersch graft from the inner aspect of the upper arm or thigh, scrotum or prepuce may be used.

FIG. 6. Spastic entropion with trichiasis.

Following the eyelid anesthesia the scar area is excised, care being taken to remove all the cicatricial tissue and to unite the



A B

Fig. 7. Birch-Hirschfeld operation. A, movable orbicularis muscle slips. B, insertion of muscle slip sutures.

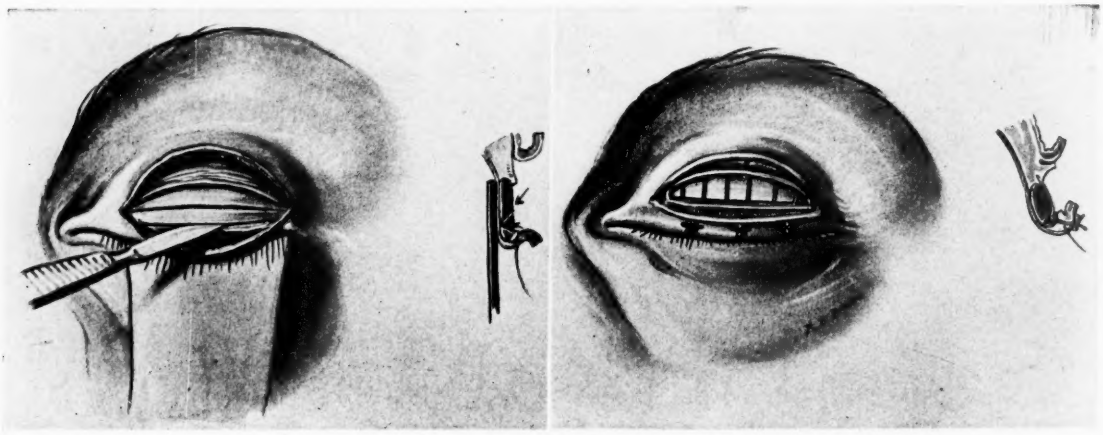
eyelid margins by two or three partial tarsorrhaphys. This opens up the excised area, giving a full sized bed for the graft and assures that the graft will be held in this position. If the skin of the upper eyelid is used, it is cut the exact size of the opening into which it is to be placed. The bed should be clean and free of bleeding points. The graft should have as little subcutaneous structure as possible and be handled without pinching with forceps. It is sutured in place by frequent interrupted No. 000 black silk sutures, with the same care as to trauma. Should a Thiersch graft be used, a slight larger graft is placed, to allow for cicatricial contraction. The dressing applied should be, first, a rubber tissue with grain running in two directions and lightly vaselined, next, an evenly applied gauze dressing and firmly applied adhesive plus a bandage, held by well placed adhesive strips. This is to spread the graft evenly and firmly against its bed, preventing all movements of parts containing the graft or immediately adjacent to it, or possible secondary hemorrhage under the graft. First dressing is done five days later.

ENTROPION

When a spastic rolling inward of the eyelid margin occurs from an irritative conjunctival condition with a spasm of the marginal orbicularis palpebrae muscle overcoming an atonic or pathologically weak orbital portion of this same muscle, or from lack of support received from the eyeball, surgery is indicated if unrelieved secondary permanent pathological changes occur which simpler measures cannot relieve. The condition is exaggerated by the continual irritative reaction due to the eyelashes' brushing against the cornea and conjunctiva.

After eyelid anesthesia, the cautery punctures as for ectropion may be done, observing the same technique only from the skin side, the needles passing through the eyelid structures just down into but not through the tarsus. Sutures similarly placed following the same steps as mentioned for ectropion, again from the skin side, may be used. For a hypertrophied, over-powerful marginal portion of the orbicularis palpebrae muscle, the Celsus operation makes an incision through the skin 3 mm. below the cilia, cutting a 3 mm. strip of the orbicularis muscle from the cilia follicles inferiorly, the length of the tarsus. The skin incision is closed by interrupted black silk sutures. Birch-Hirschfeld, instead of excision through a similar skin incision, outlines the 3 mm. strip by incisions down to the tarsus. He incises through the center and inserts a double armed suture in each cut end. The temporal

end is carried up and toward the nose, the nasal end down and temporally, catching the tarsal surface. Then it is taken out immediately; reinsert 3 mm. apart, catching topmost edge of lower tarsal cut edge, continuing onward up through lower



A B

FIG. 8. Streatfield-Snellen operation. A, third stage. Wedge-shaped excision of tarsus. B, fourth stage. Insertion of double armed sutures.

through the skin separate from the skin incision. The skin incision is closed separately by interrupted black silk sutures.

If the entropion is partial and at the outer one-third of the eyelid, the Spencer-Watson procedure relieves the rolling in and the trichiasis as well.

When the entropion is due to cicatricial contraction with tarsal deformity and trichiasis, the Streatfield-Snellen procedure is to be used following the steps indicated:

1. Insert lid plate. After an incision through the skin, 2 mm. above the lashes, parallel to and extending the length of the lid margin, the upper skin flap is undermined.

2. The orbicularis muscle is excised over the tarsus for a width of 2 mm. immediately under this skin incision.

3. A wedge of 1.5 mm. base with apex toward the conjunctiva is excised from the entire length of the tarsus, care in cutting through conjunctiva being taken.

4. Three double armed sutures are inserted as follows: through needles inserted 3 mm. apart, septum orbitale, sponeurosis of levator muscle, and partly through upper border of the tarsus, coming skin flap above lashes; tie over rubber pigs. Unite skin wound edges with interrupted skin sutures.

5. Dressing. Redressed on second day and daily thereafter. Sutures are removed on the fifth or sixth day.

Should the entropion follow a second degree burn of the lower conjunctiva with cicatricial contracture, excision of this area, followed by a mucous membrane graft from the lip or cheek, may be done with success.

SUMMARY

Surgical experience dealing with spastic ectropion or entropion where early eyelid structural changes have set in, has shown that the simpler operative procedures are quite uniformly successful; and that where permanent damage has occurred the plastic operative procedures described give excellent cosmetic and functional results.

REFERENCES

1. VON BLASKOVICS, L., and KREIKER, A. Eingriffe am Auge. Stuttgart, 1938. Ferdinand Enke Verlag.
2. THIEL, R. Atlas der Angenkraukheiten. Leipzig, 1937. Georg Thieme Verlag.
3. WEEKS, W. W. Surgery of the Eye. 1938. Blanchard press.

THE SURGICAL TECHNIQUE IN TENOTOMY OF THE INFERIOR OBLIQUE MUSCLE

JAMES WATSON WHITE, M.D.

Associate Professor of Clinical Ophthalmology, New York Post-Graduate Medical School

NEW YORK CITY

THE indications for tenotomy of the inferior oblique are first, those cases of head tilting in which the tilt serves to bring the images of the two eyes to the same level so that they may be fused. When the eyes are thus brought to a level, considerable amounts of divergent or convergent strabismus may be overcome. This was so in a case of Duane* in which there was a marked torticollis. When a collar was used to straighten the head, the eyes crossed and one eye went markedly up. Upon removal of the collar, the head again tilted to one shoulder and the eyes straightened. A like head tilt is often produced when binocular single vision is not obtained because of poor vision in one eye, or when total suppression is present. The propreceptive muscle sense is the most probable explanation of this tilting.

The deformity other than the head tilting may be a marked convergent strabismus in which the non-fixing eye turns sharply in and up. In some cases, when the right eye fixes, the left shoots up and in, but when the left eye fixes, the right eye turns markedly up and in. About the same condition is found in a divergent strabismus, the non-fixing eye being both diverged and elevated. Either of these conditions may be unilateral or bilateral. It is caused by a paralysis, more or less marked, of one or both superior rectus muscles. Fixation with the eye of the paretic muscle causes a secondary deviation of the associate muscle of the other eye, which, continued, often produces an hypertrophied muscle. When the condition is unilateral and the non paretic eye is used to fixate, no such hypertrophy develops. Hence the deformity is never so great, the paretic eye being more or less hypotropic.

A second indication for a tenotomy of the inferior oblique, described by Duane in an unpublished article, is to correct a primary spasm of the inferior oblique. This is of rare occurrence, the hypertrophy being produced, practically always, from the continuous secondary deviation present when fixing with the paretic superior rectus muscle in the field of its action as an elevator.

A third indication is found in those cases of paralysis of the superior oblique muscle in which the direct antagonist or inferior oblique of the same eye develops a secondary contracture, being more or less unopposed, depending on the degree of paralysis of the superior oblique muscle. Such cases are much less frequently seen than are those due to a paralysis of the superior rectus muscle of the contralateral eye.

The most frequent symptom is the head tilt, which, because of the malposition, may cause pain or soreness in the muscles of the neck and back. Insurmountable diplopia is quite frequently seen, due to the variability of the deviation as the gaze is shifted in different directions. Headaches, asthenopia and nervousness are also common symptoms.

Surgically, the cases arrange themselves into several groups.

1. Unilateral superior rectus paralysis, when fixation is preferred by the paretic eye.

When the deviation is over 5^{∇} and increases to 10^{∇} or more in the field of the paretic muscle, a tenotomy of the inferior oblique may safely be done. If the amount of the deviation is less than this, a resection of the paretic superior rectus is safer. If

* Fuchs's Text Book of Ophthalmology, page 308, 8th edition.

there is a secondary contracture of the inferior rectus so that the deviation is carried into the lower field, a recession or tenotomy of this muscle may have to be made, but in a subsequent operation to the tenotomy of the oblique. If, however, the non-paretic eye is used to fixate and it is decided to resect the paretic superior rectus, the opposing inferior rectus may be tenotomized at the same time.

2. Bilateral superior rectus paralysis, when the same eye is constantly the fixing eye.

Usually the more paretic eye is used to fixate in such cases. The non-fixing eye may be either divergent or convergent, in which case the external rectus is resected or receded as indicated, but while it is held on the muscle forceps after being freed from the insertion, the inferior oblique is tenotomized at its insertion. If there is no definite lateral deviation, the inferior oblique is tenotomized in the usual manner.

3. Bilateral superior rectus paralysis, when the amounts of the paralysis of the recti are about equal and fixation is shifted readily from one eye to the other.

In such cases, if the left eye shoots markedly up in looking to the right, and the right eye up in looking up to the left, a double inferior oblique tenotomy may safely be done, provided the paralysis of the superior recti is not marked. However, if the paralysis is marked, the paretic superior recti should be resected. If the upshoot is about equal but not marked and a convergent squint is present, both interni may be receded or both externi resected, at the choice of the operator. This will depend on the degree of abduction, adduction and the strength of the converging function. If, however, a divergence is present, both externi may be receded. A resection of the interni might produce an increase in the upshoot in the nasal fields.

4. When the superior recti are paretic to a different degree, it is safer to tenotomize first the inferior oblique that is the more overacting. Later the superior rectus of the lower eye may be resected or the opposite inferior oblique tenotomized, in the judgment of the surgeon.

Contraindications. When one superior rectus is almost completely paralyzed, the paretic muscle should be resected. When both superior recti are very paretic, the inferior oblique should not be tenotomized, since they are needed as elevators. In such cases, both superior recti should be resected.

The inferior oblique may be tenotomized if there is a primary spasm of the muscle. There should be no contraindication.

For a paralysis of the superior oblique, the inferior oblique may be tenotomized only when the deviation is marked in the upper field of this oblique; i.e., if the right superior oblique is paretic, the deviation is usually greatest in looking down to the left, but if the secondary contracture of the inferior oblique of the same eye causes the deviation to be almost as marked in looking up to the left as it is in looking down to the left, the right inferior oblique may be tenotomized. This is contraindicated if the deviation is definitely more marked in the field of the paretic superior oblique, in which case a satisfactory result may be had by a tenotomy or recession of the inferior rectus of the opposite eye. Wheeler,[1] described tucking the paretic superior oblique. This was done in one of my series.

The indications for the tenotomy of the inferior oblique were first described by Von Graefe, but there is no record of his having performed the operation. Dunnington[2] gives a very full history and bibliography of the operation.

Duane first performed the operation in 1906, and, after operating on eight cases, reported them. In discussion, many dire consequences were prognosticated but, in a personal communication, Duane said, "not having had any of the bad results claimed, I continued to do them." This evidenced the opposition to the procedure at that time. Such opposition still exists on the part of those who are theoretically opposed to the operation or who have operated without recognizing the contra-

indications. The writer has either operated, or advised the operation in well over 1,000 cases and the very few unsatisfactory results have been due to an underestimation of a contraindication.

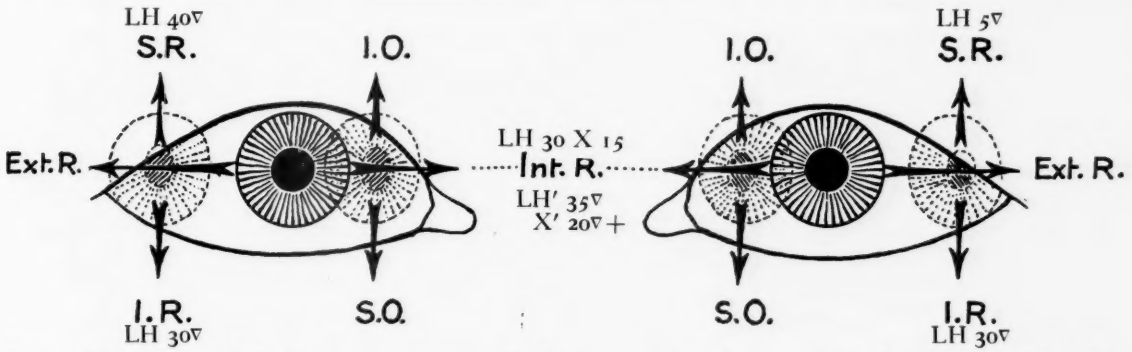


Fig. 1. LH 30▽, X 15▽ indicates the amounts of left hypertropia and exotropia at 6 m. LH' 35▽, X' 20▽ is the amount of the same at 33 cm. The amounts indicated in the upper and lower corners are taken at 33 cm.

TESTS

The most satisfactory test is the screen (cover) test with its modifications. The test must be made in the eyes front position for distance, near, and also in the six cardinal positions. In the smaller amounts of deviation, the screen-Maddox rod test is valuable provided suppression is not present and the patient is old enough to coöperate.

The screen test alone with the deviation measured accurately by prisms in all positions may be made in all cases if the patient has central fixation. If there is an associated lateral deviation, it should be corrected at the same time as the vertical by prisms held base out or in as required. The test is possible even in many children under three years. This test is followed by the screen comitance test, the results of which often give the final cue as to which operation is indicated. It is as reliable in the very young as in adults, does not depend on central fixation, and is most valuable.

These findings may be graphically recorded. (Fig. 1.) With such a record before and after operation one can readily study the several features of a case and in measuring after the operation, one can accurately judge the effect of the operation.

TECHNIQUE

The operation may be performed through a skin incision or through the conjunctiva. When it is done through a skin incision, a vertical line is dropped from the supraorbital notch to the infraorbital margin. (Fig. 2.) This point is used as the inner limit of the incision, which is carried out 5 to 8 mm., following the natural folds of the skin. In the earlier operations the incision was made much longer, but improved technique has reduced it to the length stated. At first this incision was carried through the orbicularis to the orbit margin, but when carried to this depth, hemorrhage was at times quite marked. When the incision just through the skin is followed by a blunt dissection, hemorrhage rarely occurs.

After the skin incision is made, the wound is held open by small forceps, and with Stevens scissors the fibers of the orbicularis are separated by blunt dissection. (Fig. 3.) When the orbital fascia is reached, it is picked up by the forceps just far enough above the orbital margin to allow the same blunt dissection to be continued into the orbit, after an entrance is snipped through the fascia. Through this opening a strabismus hook is introduced, usually from the temporal side with the point of the hook toward the nose. The hook is carried along the floor of the orbit, pointed at an angle of 45 degrees up and in. When the hook is drawn up into the wound it should engage the oblique muscle at right

angles. The lids are now held open and traction is made to determine whether the muscle has been properly engaged. If it has, it is brought up into the wound. (Fig. 4.)

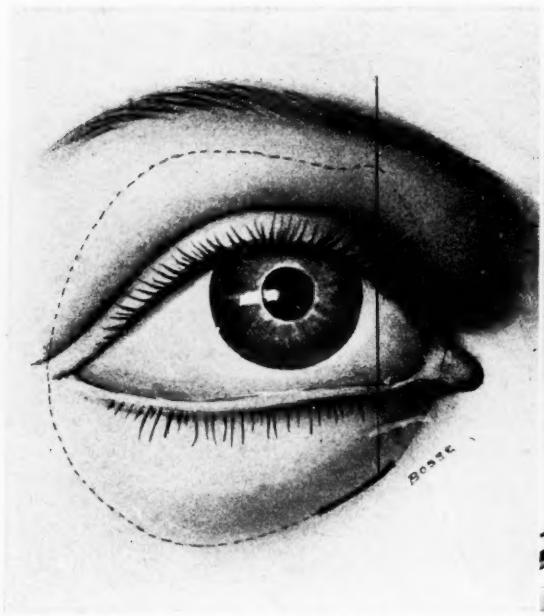


Fig. 2. Showing a line dropped from the supraorbital notch to the infraorbital margin and its relation to the skin incision. (From White, in *Tr. Am. Opbtb. Soc.*, 1933.)

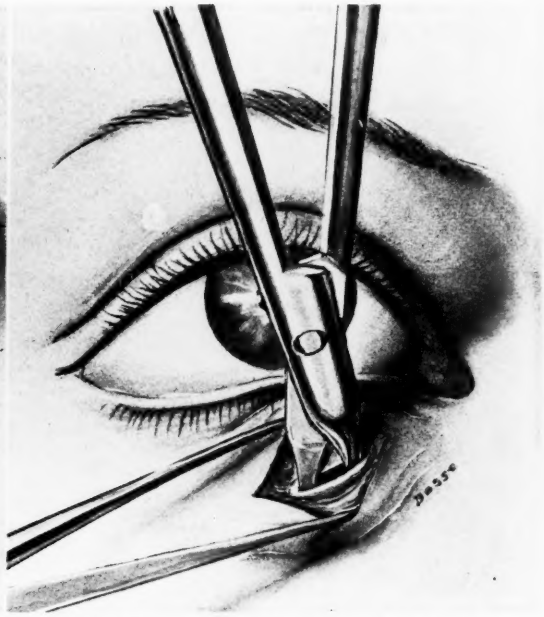


Fig. 3. Blunt dissection through the orbicularis muscle and the orbital fascia. (From White, in *Tr. Am. Opbtb. Soc.*, 1933.)

Usually some fibers of the orbicularis cover the tendon, but when these are dissected away, the white glistening tendon of the oblique is seen on the hook. The tendon is stripped clean in both directions and the loop of tendon on the hook is grasped by mosquito forceps or fixation forceps with a catch. (Fig. 4.) The hook is left in place. The tendon is then cut through from 3 to 5 mm. on either side of the forceps, thus removing a piece from 6 to 10 mm. in length. (Fig. 5.)

Occasionally, when there is difficulty in engaging the muscle, the hook may be entered from the nasal side, and directed 45 degrees down and out. After the tenectomy, the hook is reëntered and swept across the floor of the orbit, to determine whether any lateral strands remain.

The skin wound is then closed by sutures and a dressing is applied. On the following day, when there should be no hemorrhage or oozing from the wound, an extremely fine layer of cotton is placed over the incision and sealed with collodion. When this is removed on the fourth or fifth day, the sutures may be cut as the dressing makes tension on them and both dressing and sutures come off in one piece.

At first it was the practice to perform a simple tenotomy in all cases, but some lost part of the effect originally obtained by the operation. Since 1915 a piece has been taken out as described, except in a few cases where the deviation was less than 5^{v}, and the upshoot was not very marked. In such cases a simple tenotomy may be used. In two cases in which a slight effect was desired, the sheath of the muscle was cut longitudinally and the muscle fibers were dissected from the sheath; 1 cm. of muscle was resected, leaving the sheath intact. (Fig. 6.) Two cases are not enough on which to base an opinion, but the procedure seems to be logical, and in these instances the results were satisfactory.

The muscle may be approached through the lower fornix in a line corresponding with the usual skin incision. (Fig. 2.) Blunt

dissection is carried outward to the orbital margin and the muscle engaged in the same manner as by the skin route. This method is somewhat more difficult but has the advantage of leaving no visible scar.

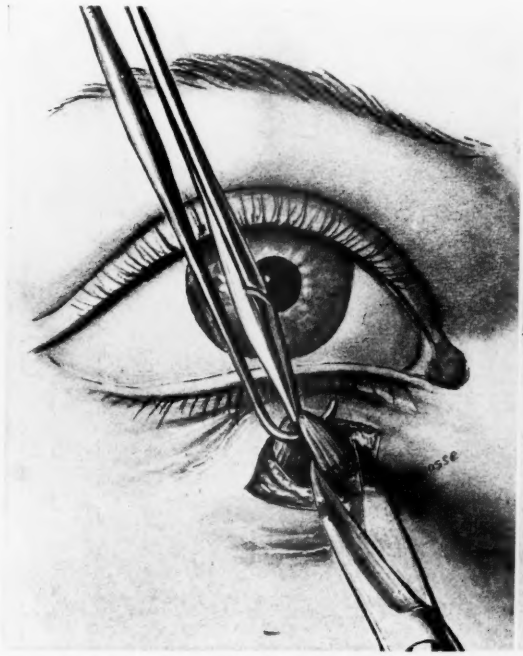


Fig. 4. The muscle has been brought into the wound, grasped by mosquito forceps, and severed 3 to 5 mm. above the forceps. (From White, in *Tr. Am. Opbtb. Soc.*, 1933.)

Fig. 5. Muscle being severed 3 to 5 mm. above the forceps. (From White, in *Tr. Am. Opbtb. Soc.*, 1933.)

One inexperienced in the tenotomy of the inferior oblique when done through the skin or by the lower fornix route may easily miss the muscle entirely. This results from not properly entering the orbital fascia.

Biopsy has shown the resected tissue to be sometimes entirely muscle, and sometimes both tendon and muscle. This is of no consequence.

Since these cases are so frequently associated with a convergent or a divergent strabismus, it seemed logical, if a resection or recession of the external rectus was contemplated, to tenotomize the inferior oblique at its insertion. This has been done for some years by several operators. After the externus is grasped by a muscle forceps and tenotomized, the muscle is raised and carried away from the globe. The inferior oblique muscle may be found directly in the field, or if not, can readily be engaged by a fine muscle hook.

After the muscle has been tenotomized, the externus is resected in cases of convergent strabismus or receded if a divergent squint is present, provided it is of a divergence excess type.

The approach to the muscle at the insertion as described has been found so satisfactory that the inferior oblique has been approached at the insertion directly. An incision is made diagonally down and out, beginning 5 mm. outside and below the external rectus insertion and carried to the heavier tissues at the fornix.

Enough cases have not been operated by this approach to report conclusions, but judging from my own results and those of confrères to whom this approach was described, one is inclined to believe that the method should be continued. The general consensus of opinion is that the muscle is much more readily engaged by this route.

If, with the paretic superior rectus, there is a secondary contracture of the inferior rectus, this muscle may have to be receded or tenotomized. It is rarely advisable to

operate on both muscles at the same time as is shown in the following case.

Mrs. M., following an automobile accident

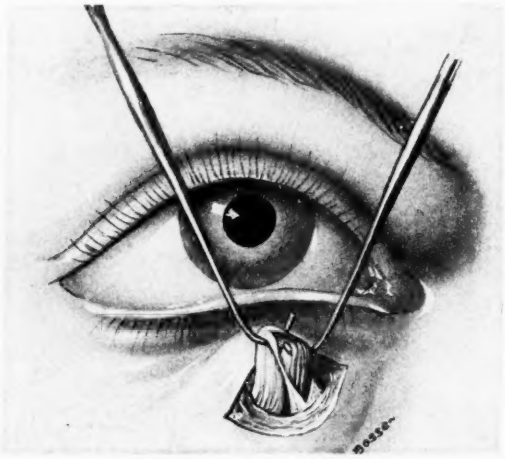


Fig. 6. While the muscle is held on a hook a longitudinal incision is made through the muscle sheath and the muscle fibers are separated from the sheath; 5 to 10 mm. of the muscle fibers are then removed. More or less may be excised as the case requires. (From White, in *Tr. Am. Opbth. Soc.*, 1933.)

three years before, had a vertical and crossed diplopia unless the head were tilted to the left. In the primary position, the left hypertropia was 28$^{\nabla}$ which increased to 30$^{\nabla}$ in eyes up and right, and 30$^{\nabla}$ in eyes down and right. In looking to the left and up, or left and down, there was but slight if any diplopia, varying from single vision to a slight double vision.

Tenotomy of the left inferior oblique at the insertion reduced the hypertropia from 28$^{\nabla}$ to 20$^{\nabla}$ in the primary position. Measured while on the table, the right upper field was corrected quite satisfactorily, but the left hypertropia in looking down to the right was unchanged. The day following the operation, she returned to her home in a distant city and to the care of her ophthalmologist, with instructions to return in six weeks for the final operation. On her return, the left hypertropia had reduced from 20$^{\nabla}$ to 10$^{\nabla}$.

With much less head tilt she could easily maintain single binocular vision. At this time, the right inferior rectus was tenotomized. This resulted in less than 1$^{\nabla}$ of left hypertropia, with the head straight. The head tilt, headaches, asthenopia, neck and backache were entirely relieved.

Similar cases were described in a thesis,[3] as belonging to group III. Patients in this group, with few exceptions, require either a recession or tenotomy of the overacting inferior rectus, but this should be postponed as long as satisfactory improvement follows the first operation. In the above case if one had at once operated for the 20$^{\nabla}$ of deviation remaining after the inferior oblique tenotomy, there would probably have been an overcorrection of about 10$^{\nabla}$, or the amount of improvement in the six weeks' absence.

CONCLUSIONS

1. Tenotomy of the inferior oblique is a most satisfactory operation when indicated.
2. The indications and contraindications should be carefully considered.
3. The most satisfactory means of study is by the screen (cover) test and its modifications. By this test only can one evaluate the degree of paralysis, of primary deviation, of secondary deviation and of secondary contracture.
4. The fixing eye should be definitely determined in an actual tropia or the dominant eye in cases of phoria.

REFERENCES

1. Wheeler, J. M. Advancement of superior oblique and inferior oblique ocular muscles. *Am. J. Opbtb.*, 18: 1 (Jan.) 1935.
2. Dunnington, J. H. *Tr. Am. Opbtb. Soc.*, 27: 277, 1929.
3. White, J. W. *Tr. Am. Opbtb. Soc.*, 1933.

A REVIEW OF SOME MODERN METHODS FOR OPHTHALMIC PLASTIC SURGERY

EDMUND B. SPAETH, M.D.

Associate Professor of Ophthalmology, University of Pennsylvania Graduate School of Medicine

PHILADELPHIA, PENNSYLVANIA

A HISTORICAL review of this rather specialized branch of ophthalmic surgery, if carried out in detail, would be most interesting. Monographs have appeared from time to time calling our attention to the antiquity of many procedures which have continued, even in their original form, from the earliest days of surgery. Nasal plastics were done by Hindu and by Arabian doctors long before the time of antiseptic and the later aseptic surgery. There is strong evidence to suggest that bone plastic surgery was also done at an early period of time. Soft tissue pedicle flaps were common with several of the early central European and Italian surgeons, and their procedures continue in part with but few modifications, up to the present time. The injuries connected with warfare compelled the ophthalmologist of the generation which is now passing to devise and to invent flaps and grafts, and even new dressings, to such a degree that modern ophthalmic plastic surgery, as a specialty, was born through them. The World War demanded that they utilize their ingenuity to a degree previously uncalled for. Modern life, the traumatisms connected with the industries, automobile and aeroplane accidents, and even accidents in the home, are now an ever increasing source for cases needing reparative surgery.

It is, as Wilmer said, in the foreword of Spaeth's "Ophthalmic Plastic Surgery," "The eye possesses certain tissues which exist in other parts of the body, but it is also endowed with some histologic elements which are peculiar to its adjacent structure and which are formed most specifically for their particular function. The restoration of these parts requires special knowledge and training, and therefore, belongs logically to the domain of ophthalmic surgery. No argument is required in order to convince the most casual observer that the destruction of sight is a most irreparable injury, but the tragedy that may lie in a distorted physiognomy is not so readily understood. Only one who realizes the mind's construction in the face can consider the secondary physiological effects upon the face. To reconstruct such a deformity is to reconstruct the man by relieving him of the 'one auspicious and one drooping eye,' as Shakespeare expressed it. No rehabilitation is more important or far reaching in its results."

Ophthalmic plastic surgery, however, is even more far reaching than that alone connected with trauma. Ptosis of a lid, other congenital defects, such as epicanthus and ankyloblepharon, benign and malignant neoplasms, such as neurofibromata, hemangiomata and retrobulbar sarcomata, demand a detailed knowledge of plastic surgery for a satisfactory correction. The surgery for a symblepharon, as, for example, following an alkali burn of the conjunctiva, embodies definite principles, as does the reconstruction of a lower lid destroyed by an automobile accident. One hears an ophthalmologist occasionally state a dislike for plastic cases though his next operation may be an exquisite plastic repair, with the conjunctiva, of a ruptured cataract incision. Such a person is as capable of doing a reconstruction of the socket or of implanting a cartilage graft into the orbit as he was with the conjunctival plastic operation mentioned.

In the correction of soft tissue defects there is one basic principle which covers all cases. It is that "tissue which has been lost must be replaced." An extensive

cicatricial deformity may seem upon casual examination to be due to the loss of a great amount of soft tissue. If one can, however, in such an instance, visualize or imagine the resection of that scar present and will, in his mind's eye, fit the resulting notches, pedicles, and triangles into an original pattern from whence they came; then he can, also, decide immediately whether the defect in question is due wholly to malposition of the tissue consequent to healing, or whether it is due to an actual loss of soft tissues. In the former instance, it is simply a matter of scar resection and of subsequent suture. In the latter, however, either a flap or a graft of some type must be used.

Fig. 1. Preoperative sketch of incision and suture lines.

The most difficult part of plastic surgery is that done upon a drawing board before the patient even reaches the operating room. It is the careful planning necessary to obtain maximum success. Photographs should be taken and sketches must be made to show the original defect. Cellophane can be molded directly to the patient's face and the defect traced on it with a red wax pencil. With the photographs and sketches on a drawing board, all necessary scar resections may be planned. The amount of soft tissue which has been lost can be determined and one can decide the source from which this is to be obtained for replacement. Figure 1 illustrates such sketches made preoperatively and should be compared with the incision and suture lines actually obtained through the surgery. An ectropion, however, which illustrates without the slightest doubt that soft tissue has been lost must have that replaced. The same reasoning would apply to a bony defect. One can be most certain that a cartilage graft is necessary to elevate an eyeball to the same level as its mate. Such sketches can then be taken into the operating room and in all except most unusual cases, the plan outlined should be carried out.

Skin Grafts. An epidermal graft, in its process of "taking" is undoubtedly kept alive by the early formation of capillary loops from the "bed" into which it has been forced and held by the pressure of the dressing. Fibrin may also play a part in this process of healing. Blood serum, however, if it collects or stands beneath the graft is perhaps the cause for most failures, when using a free skin graft. A dermo-epidermal graft, as placed into position, receives necessary nourishment from the edges of its reception "bed" as well as from the healthy fixed tissues in contact with its under surface. In all such cases there is some slight round cell infiltration, and some fibrin is present. The capillary loops increase to a very great degree, penetrating the derm of the graft. Superficial desquamation develops in the uppermost cells of the graft. Here, as well, a failure will result if a hemorrhage occurs later beneath the graft. Another important point is to prevent the formation of unfilled spaces beneath the graft or flap. Such spaces are more likely to occur with dermo-epidermal grafts, but

prevention is equally important in the use of cartilage and bone.

Success with an epidermal or dermo-epidermal isograft is uncommon. These grafts have perhaps tissue reactions rather similar to those which make imperative the detailed typing and matching of donor and of recipient for blood transfusions. Many other authors condemn them as quite valueless, though Elschnig reported a successful take in the case of a sister to sister. Formalized cartilage remains, when embedded, though perhaps only as an encapsulated foreign body. Magitot has shown that after some time this becomes infiltrated with penetrating bands of scar tissue and that in spite of some very slight, but perhaps inevitable loss, the major portion of the graft remains permanently intact. Isografts of cartilage have been successful repeatedly. It is quite possible that the statement relative to formalized cartilage also applies to human cartilage isografts. The author has had no experience with isografts of bone.

Grafts and flaps must never be pinched. A free skin graft should be removed from the surface of the razor blade and placed directly on the site prepared for it, or wrapped about the necessary mould without delay, if this is possible. It is not necessary to place the graft in warm saline unless unavoidable delay arises in its use. A dermo-epidermal graft or a flap can be lifted and moved about by one or more sutures placed through its edge even before it has been freed from its original position. These sutures may also be used for traction to assist in cutting the graft. Cartilage and bone grafts may be wrapped in warm saline-soaked gauze sponges for any necessary handling or because of delay in embedding them. Warm vaseline, applied lightly to the skin surface as well as to the side of the warmed razor blade, assists appreciably in cutting epidermal grafts.

Two small sharp hooks, attached to each end of a stout 4-inch chain, are quite satisfactory for holding the skin taut while the graft is being cut. Two pairs of such hooks are necessary, the operator holds one pair while the other pair is held by the assistant. Tongue blades are almost equally satisfactory. Grafts should be cut, if possible, without perforations. This is especially important in socket repairs. When such perforations occur, however, the hole may be capped by an overlying additional strip of epidermal graft. An epidermal graft has an immediate contraction of somewhat less than one-half its plane surface, a dermo-epidermal graft slightly less than this.

There are two basic methods for the utilization of free skin grafts, whether dermo-epidermal (Wolfe) or epidermal (Ollier-Thiersch). Either they are placed flat into the defect (after the dissection is prepared for them), or they are wrapped about a mould and buried into the defect (Knapp, Esser, and Gillies). The former is by far the more common with dermo-epidermal grafts; also sutures are necessary with this latter type of grafting. Epithelium must not be grafted upon the bulbar conjunctiva as the natural desquamations which form may cause a chronic mechanical conjunctivitis. Naturally, this does not apply if the eye is lost as an organ of vision. Under such conditions, an epidermal graft wrapped over a metal conformer is an ideal method for correcting the symblepharon. The prosthesis can be worn applied in close contact to the blind eyeball. The inferior cul-de-sac, however, can tolerate fairly well epidermal grafts for a cicatricial entropion or for symblepharon, as long as the epithelium is not juxtacorneal.

The first essential is the resection of all scar tissue, both buried masses and bands, as well as the more evident superficial portions. Assuming the correction of a case of ectropion with a dermo-epidermal graft —the dissection for its reception is completed, the graft is lifted from its original bed, transferred to its new site and then sutured directly into position. The best example of this is the use of a free skin graft from the opposite lid. The method applies very nicely to the correction of soft

tissue loss and ectropion without much cicatrix, in the repair of cicatricial incisurae, or because of a notching of the lid margin.

FIG. 2. A, the correction of ectropion by Gillies technique. B, correction at the time of removal of the mould. C and D, another patient, shown before and after correction of the defect. In C, the patient is making a strong effort to open the lids.

A deformity near the outer canthal angle also responds well to this procedure.

The correction of a cicatricial ectropion with an epidermal graft is best illustrated by Wheeler's method, especially in a case where the lid margin may be drawn to the level of the eyebrow. In this the scar is resected by incision, the incision spread by blunt dissection along the natural lines of cleavage, the lid margins sutured together to form permanent intermarginal adhesions, and the graft laid, in one piece, over the defect formed by the dissection. To obtain a permanent tarsorrhaphy, two small areas on each of the lid margins, lying exactly opposite to each other and immediately posterior to the line of lashes, each at the junction of the middle and inner and middle and outer thirds on the lid margins, are notched and denuded for a distance of 3 mm. and for a depth of 0.5 mm. Silk sutures are mattressed through these opposing notches and tied over tiny rubber plates. Healing will result in a firm adhesion between the two lids. These intermarginal adhesions are permitted to remain intact from three months to a year while massage is being applied to the reconstructed lids to prevent further cicatricial contraction.

The correction of an ectropion of one lid alone, i.e., where both upper and lower lids are not involved, is best carried out by the Gillies inlay method (Hess and Esser principles) a procedure which he (Gillies) outlined during the late war, and which still stands as a most satisfactory method of treatment. In this the scar is first resected. A mould of dental stent is then made, of such a size that it will fill the defect and still allow its edges to be coapted and closed by a line of sutures. The epidermal graft is wrapped about this mould, epithelial surface innermost, and the mould with the graft about it is sutured into position. At the time of the first dressing, this line of sutures is removed, the graft as it lies upon the mould is incised, the mould is removed and the epithelialized pocket opens outwardly to supply the correction desired. (Fig. 2.) Many cases of simple noncicatricial ectropion of the lower lid respond well to the basic techniques of Kuhnt, of Blascovicz, and the more recent operation of Vogt, in that a shortening of the arc of the orbicularis fibers, plus the removal of the redundant conjunctival surface is adequate for full correction.

This same method is ideal for the correction of a contracted socket (Knapp and Wheeler methods). In such cases any soft tissue replacement necessary for the lid margins or of the lid epithelium itself should be done first (Fricke's principle). Because of the more or less marked conjunctival deficiency present, it may be necessary to use pedicle flaps in these instances. When the patient is ready for the socket correction, the line of the palpebral fissure on the lid margins is incised posterior to the lashes and all cicatrix resected from within the socket. The incision line should pass behind the lower lid to the level of the bony orbit. After incising this, the dissection is to be carried upward and backward, but well away from the upper rim of the bony orbit to save the orbicularis and intact fibers of the levator palpebrae superioris. The socket is fitted with a mould of dental stent, the graft wrapped about it, mould and graft placed in position, and the lid margins sutured together with temporary sutures. This mould must fit snugly, but without undue tension, to the lids, into the entire bed prepared for it.

Entropion, is not so readily corrected, though mucous membrane grafts (van Milligan) are valuable in minimal conditions, as also in those of greatest degree wherein (Spaeth) a complete cul-de-sac restoration is necessary. The operations of Snellen, of Hotz, and of Beard are all important contributions.

The above conditions illustrate the majority of the important indications for the use of both types of free skin grafts. As a recapitulation, they are to be used only in those cases wherein there is a definite loss of soft tissue, and the rather definite probability of removing all scar tissue deposited in the defect. This includes a large portion of the cases which appear for correction, but it does not include many more serious and more difficult cases: those with extensive loss of soft tissue; those of long standing with a large amount of cicatricial contracture which cannot be wholly and cleanly removed; those without a firm base or a firm layer of normal tissue upon which the free skin grafts can be satisfactorily placed; and those cases wherein the tissues contiguous to the defect are themselves contaminated with contracting cicatrices. This last type of case, incidentally, is the one positive indication for the use of delayed pedicle flaps: skin flaps from the neck and chest by means of long cervical pedicles. These may be transferred and shifted two or three times in moving them from their original site to the correcting position.

Pedicle Flaps. Cutting a flap forms, o necessity, new and additional scar lines. This is a factor one must consider when the patient is young and a female. The edges should be cleanly cut, though beveled if possible (Hunt), and any subepithelial fat which is adherent removed from their posterior surface. A suture placed in the apex is adequate as a tractor for moving the flap about without damaging it with forceps. It is necessary to allow for a slight contraction in the size of the usual pedicle flap though this is much less than the one-third to one-half contraction of the plane surface allowed for with free skin grafts. As many sutures as are necessary may be used, but they should be placed with very small bites and the finest possible suture material and needles used. A flap placed in position under tension is foredoomed to failure. It should lie smoothly but should not be stretched. Anchorage sutures, when necessary, are to be buried and should be of double o, or even triple o, plain catgut.

The flaps ought to be cut so that their long axes parallel the normal tension lines of the skin as they lie about the orbit, the lids and over the malar and zygomatic processes (Hunt). These tension lines follow the direction of the muscle action of all the muscles, and the tension of the ligaments which lie under the skin in this region. Those we must consider are: the ascending forehead fibers of the occipitofrontalis; the circular fibers of the orbicularis oculi; the pull of the two canthal ligaments, and the horizontal raphé-like action of the fibers

of the orbicularis especially at the inner canthus; the pyramidalis nasi and its oblique fibers lying over the bridge of the nose; the horizontal fibers of the compressor nasi; lower down, the caput angulare, the caput infraorbitale, and the caput zygomaticus of the quadratus labialis superior; the caninus, the zygomaticus major, and especially the obliquely descending pull of the large masseter and the temporal muscles. Even the anterior aricular muscle is a factor in this matter of tension lines. They are not to be confused with the histologic tension lines which lie in the skin about the face for these have no relationship to the direction of muscle pull beneath it (Lange). Unnatural, strange, and new expressions can be obviated if care is taken in locating the direction of the different flaps.

Finger-like pedicle flaps from above the eyebrow (Fricke), using the skin of the forehead or the hair-free skin over the temples, are usually quite satisfactory for the correction of many cases of cicatricial contracture, and especially for contraction ectropion of the lower lid where there has been a loss of considerable soft tissue. These same flaps may be raised with a pedicle attachment at each extremity (Axenfeld, Kuhnt), when it is necessary that they be of a considerable linear, or lengthwise, size. The central portion of such a hammock flap is moved downward into its correcting position. After healing has occurred, these two pedicles may be returned or resected and discarded. Such flaps have undoubtedly a greater margin of viability, but their use is rather limited.

Figure 3 illustrates the complete loss of the upper and lower lid following complications which occurred with the reconstruction of a contracted orbit. Socket repair was satisfactory, though a pressure lid sloughing occurred. A pedicle flap was brought down from the temporal region to fill in necessary tissue at the site of the lower lid. The margins of those lid remnants remaining were then opened and a second flap turned in upon itself, rather like a hinged door. This was sutured to the posterior lip of the freed lid edges of the remains of the upper and the lower lids. The raw anterior surface was covered with a free skin graft. The repair was completed by opening this curtain of soft tissue horizontally as the new palpebral fissure. The patient is awaiting, at this writing, a hair-line graft on the upper lid from the opposite side to replace the lashes. She is wearing a glass conformer in lieu of a glass eye in the photograph.

A B C

Fig. 3. Reconstruction of both upper and lower lids. In c, the patient is wearing a silver conformer, while awaiting a permanent glass prosthesis.

Morax combines pedicle flaps very nicely with free skin grafts for the repair of a contracted socket, especially that type of contraction and deformity found after evisceration. The lids are reopened, turned upon themselves, their posterior surface grafted and after this "take," they are returned to their original position over a temporary mould, this to be replaced later by a prosthesis. This principle was that used in the correction illustrated in Figure 3.

Figure 4 shows a canthal deformity remaining after the repair of the socket. It was corrected by means of reflecting upon

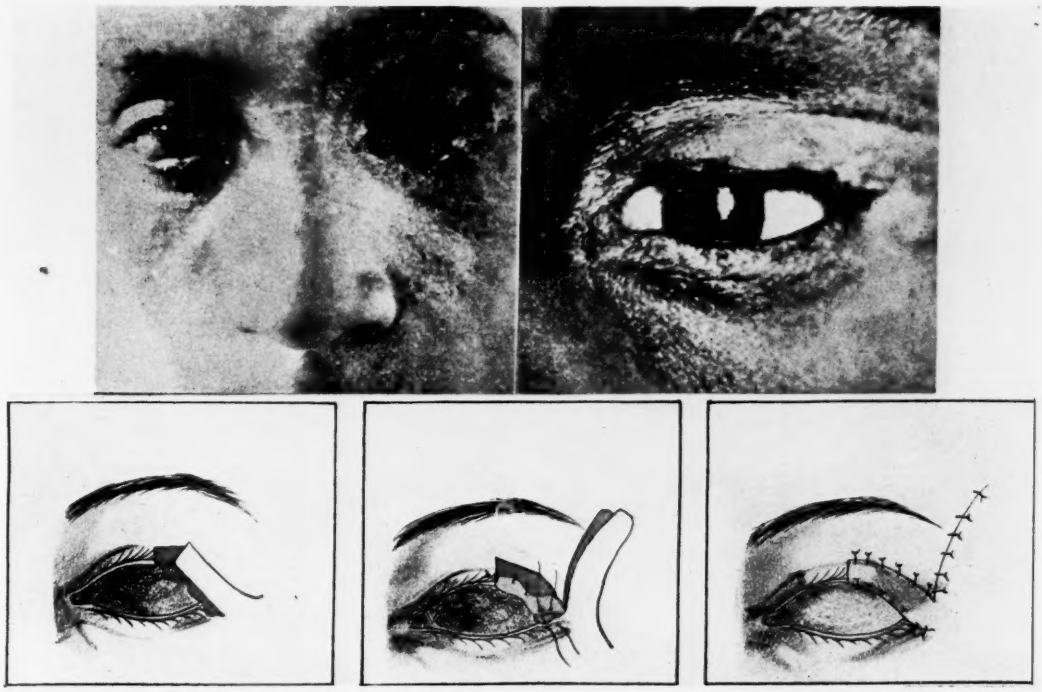


Fig. 4. The correction of a canthal deformity by means of superimposed pedicle flaps.

itself a small flap from the epithelial surface. The raw surface of the defect and of the reflected flap was covered by a small pedicle flap cut near the outer extremity of the eyebrow. The correction obtained is well seen (Kuhnt, Axenfeld, Fricke). The patient is wearing his permament prosthesis.

In this individual, even though the contracted socket followed exenteration for malignancy, an Ollier-Thiersch graft was used for the correction. Usually, however, the procedure just discussed, by Morax, or a modification of it, is more successful and certainly less difficult. The pure razor cut epidermal graft does not take well when applied to the non-elastic scar tissue covered bone and periosteum which results following late after an orbital exenteration. Skin flaps may be moved in from the side (Knapp, Celsus, Blascovicz), from above, from below, and from the nasal angle of the palpebral fissure, depending upon the defect and the degree of correction which is necessary. Some of the various flap procedures devised for the correction of an angle defect are most ingenious and are equally valuable. The simple Fuchs canthoplasty, and the author's modification of it, the Snellen and Wheeler angle tarsorrhaphies, and the Hasner, Kuhnt, and Richet canthus reconstructions are illustrative of methods which continue today as invaluable as when they were first presented.

Wheeler presented a most ingenious method for correcting lid colobomata in the central portion of the lid margin (halving operation). In this procedure the conjunctival and skin surfaces of the lids are separated, the tarsal plate is found, and separately its margins are approximated. The conjunctival surface-tarsal-plate defect is first closed with very fine sutures. Catgut must be used because these sutures are buried. A small rectangular skin flap is then dissected from the edge of the skin coloboma nearest to the line of tarsal sutures, so that, as the contralateral lip is lifted and moved laterally across the catgut sutures, it will fit into a bed pre-

pared for it upon the opposite side. The two lines of sutures, conjunctival and epidermal, are thus offset, one from the other.

Fig. 5. Snydacker technique, using a cervical flap for the correction of a gross eyelid deformity. The lower right shows a similar flap split for an upper and a lower lid.

The skin sutures may be tied over tiny rubber plates. An external canthotomy is usually necessary when using this procedure.

Figure 5 shows the use of a pedicle flap from the cervical region according to the procedure of Snydacker, as modified by Morax. In this the pedicle was first lifted and tubed. Ten days later the flap was lifted and sutured into position, and ten days after that the pedicle was resected and returned. Ten days later it was necessary to elevate the outer horn of the flap to give it further additional lift. The case was one of a healed traumatic osteomyelitis wherein the orbital floor had first been repaired by a cartilage graft.

Eyebrows may be replaced by one of several methods; a pedicle flap or a free skin graft from the opposite eyebrow; a pedicle flap from over the hair line at the temple; or best of all, by a free skin graft from the occipital scalp. These occipital scalp grafts are rather thick and while being trimmed, before they are placed into a correcting position, it is most important not to injure the hair follicles as they appear upon the reverse surface of the graft. These grafts should have their edges cut obliquely so that the posterior surface of the graft has the greater plane surface. When transplanted, the lower end of the graft should lie farthest toward the temple so that the direction of the hair is outward. Transplantation of the line of lashes is similarly done. Eyelashes also can be replaced by a free skin graft from the lower edge of the normal eyebrow.

Mucous membrane grafts (van Milligan) may be mentioned here as they apply to the repair of entropion and of symblepharon. The natural moist condition of mucous membrane permits its transplantation upon either the bulbar or the palpebral conjunctiva. Such grafts must be trimmed very thin, however, and applied snugly

with sutures. Mucous membrane does not adjust itself well to correction when wrapped over a mould or upon a conformer. Epithelium should never be used to replace bulbar conjunctiva if the eyeball is still in place. This is without exception in the case of the conjunctival surface of the upper lid and of the bulbar conjunctiva when contiguous to the limbus. The lower cul-de-sac tolerates an epithelial graft fairly well as long as one can retain a small bridge of conjunctiva next to the limbus, so that the epithelium will not be in contact with the cornea. Mucous membrane grafts can also be buried in the skin of the forehead or the skin of the face. A pedicle flap is then subsequently lifted with this graft on its posterior surface, for the reconstruction of a totally lost upper or lower lid, or both, in those cases where the eyeball is still in place. If the eyeball has been lost through an earlier enucleation, it is easier and quite proper to use epithelium instead of mucous membrane. This simultaneous reconstruction of an upper and a lower lid is always a difficult problem. Pedicle flaps with previously formed epithelium or mucous membrane lined posterior surfaces (by arranging the epithelial lined pocket upon the face or the forehead, the anterior surface of this forming the connecting head of the subsequent pedicle flap), are usually a necessary and perhaps the only certain procedure available. Axenfeld and, more recently, Hughes have advanced a technique for this correction which is quite satisfactory. These consist of any type of dissection which will permit a complete linear tarsorrhaphy; replacement of the lost tissues of the lids, either above or below the closed palpebral fissure; replacement of the hair line of the upper lid; reopening the palpebral fissure, by forming a new one at the proper level; and lastly, any necessary reconstruction work to the cul-de-sacs, especially the inferior.

Imre has developed a beautiful technique for sliding flaps. It is quite applicable to all types of defects, but especially satisfactory in the correction of defects from neoplasms. In restorations, and after the resection of an epithelioma, the flap is outlined, the necessary undermining carried out and small Burow's triangles planned so that the resection and closure will move the flap up, toward and into the defect. This method has been utilized in many different ways (Dieffenbach technique). Imre has used it to cover fistulae wherein a second buried flap of fascia had been transplanted to form the necessary base for the more superficial dermo-epidermal graph or flap (Blascovicz modification). It is the most satisfactory method for moving contiguous skin, often in large amounts, into a correcting position to overcome soft tissue loss.

Cartilage. When cartilage or bone is necessary for the correction of a defect in the bony tissues, clean dissection is the first essential. It is rather important that a layer of fascia and muscle tissue should overlie this type of graft. The line of skin sutures or of pedicle flap sutures must not lie immediately above the graft. Failure from secondary infection is quite likely if this cannot be arranged or is disregarded.

The cartilage, when used, should be resected from the seventh and eighth ribs at the sternal junction of these ribs and usually from the right side. It is wise, before a rib resection is done, to make a careful chest examination, to prevent the development of complications from an old, undiagnosed, chronic adherent pleuritis. Such an eventuality has happened to the author; naturally the rib resection resulted in a perforation into the pleural cavity. In this instance, no disturbing sequelae occurred, but the accident is rather unpleasant and unnecessary.

The rib is outlined through blunt dissection, the anterior, superior and posterior surfaces all being well freed. The graft can then be outlined with a small scalpel, permitting a narrow bridge to remain on the inferior edge of the rib and in this manner preserving the intercostal nerves and blood vessels. Healing also is hastened by this strip of intact perichondrium. The outlined rib section is then cut free with a

costotome or with cartilage chisels and the cartilage trimmed to the shape and size needed. All available perichondrium must be conserved. This can be done easily if care is taken.

Bone grafts, when used, should be obtained either from the anterior superior spine of the ilium, with the periosteum intact, or as scales of bone still adherent to the periosteum, from the anterior-lateral aspect of the tibia. In the former a small saw and chisel are necessary, and in the latter a broad chisel and a mallet.

The outer bony angle of the orbit can be repaired by means of a V-shaped cartilage graft, and the rim of the orbit by a cartilage wedge, inserted under the periosteum of the bone fragments still present; in addition the use of a pedicle flap is advisable to correct the deformity of the soft tissue and buried white silk sutures (after Blair) to improve the facial paralysis.

In the correction of ptosis there are three sound procedures at our command: the utilization of the occipitofrontalis (Hess, Pagenstecker, Snellen, Derby, Hunt, Tausberg); second, that of the levator (Blascovicz, Eversbush); and third, the use of the superior rectus (Parinaud, Haab, Motais, Shoemaker, Kirby). In the first of these procedures one is transferring to the occipitofrontalis an artificial function, but it is occasionally the only method available. If the levator is to be utilized, either through resection or by a tucking, it stands to reason that there should be some function still present in this muscle. Insofar as the superior rectus is concerned, its utilization demands a normally functioning muscle and this is a circumstance not always present in ptosis, especially the congenital form. Also, the operation if done upon one eye only may cause a hypophoria; hence the procedure is recommended for bilateral ptosis only. Each of the three techniques mentioned above, however, have optimum indications and if these are adhered to the results obtained should be good.

Fig. 6. The use of crutch glasses for inoperable ptosis.

Another principle is that of mechanical support for inoperable cases. Figure 6 shows a bilateral ptosis with complete external ophthalmoplegia. Operation is quite out of the question for that would endanger the cornea through the continued exposure. The cosmetic result is quite satisfactory through the use of crutch spectacles. In these, elevating loops are attached to the upper rim of the spectacles

of such a size and height that the lid is elevated without any uncomfortable pressure while the glasses are being worn.

Entropion with ptosis, such as one sees late in trachoma, needs the removal of the tarsal plate conjunctival cul-de-sac for satisfactory correction. The operation itself is also indicated as a therapeutic procedure for trachomatous panus. Kuhnt, Wheeler, and Barrada have all emphasized the value of this practice, each separately having presented most valuable techniques.

Buried white silk sutures have been repeatedly used for the correction of ptosis, for lagophthalmos in old facial paralysis, and with obliquity of the palpebral fissure (Axenfeld, Blair, Verhoeff). Lagophthalmos when non-cicatricial, is well corrected by a similar suture.

A high degree of toxic exophthalmos with impending prolapse of the eyeball anterior to the lid margins can be held usually most satisfactorily. In the looped suture of Axenfeld, the upper end of a white silk suture, inserted primarily through the internal canthal ligament, is passed along through the upper lid parallel to the margin, and the lower end in the same manner throughout the lower lid. At the outer canthus the ends are crossed and buried after being tied with sufficient tension to obtain necessary correction. Strips of fascia lata can also be used (Blair) for similar conditions.

The *postoperative care* in all plastic correction cases is most important. Free skin grafts should be dressed with sufficient pressure to hold them firmly in place and to prevent any later crawling or wrinkling of the grafts. The inlay grafts may be covered with overlapping oblique strips of oiled silk, laid down rather like shingles on a roof, and with a sea sponge applied directly above the oiled silk. The bandage, through this sea sponge, furnishes the necessary pressure for the grafts. Both eyes are to be covered and the patient kept in bed until the first dressing is done at the end of from eight to ten days.

Free skin grafts, both dermo-epidermal as well as epidermal (as in Wheeler's method), can be covered in this same manner, or even better, with a thin wafer of dental wax or dental stent, molded over the defect before the graft has been placed (or even cut)—this to form the base for the overlying sea sponge. Flaps should have a moderate amount of pressure as well, using the strips of silk and the sea sponge, but they are to be dressed for the first time on the fourth day. At this time, one may remove each alternate suture, continuing this with every change of dressing, at forty-eight hours' interval. Pressure applied must be so arranged that the nutrient pedicle is not embarrassed.

The pressure which is applied to a dressing for the free skin graft repair of a socket must be well gauged, because sloughing of the lids has occurred in such instances. Here also, the complaint of pain is the only warning that things are not satisfactory. In only one instance in the author's experience was this matter of pain disregarded. In this case a slough had occurred and was found at the time of the first dressing.

Infection in a graft or a flap is usually accompanied by pain and by a rise in temperature—in fact, the patient may become quite ill. In many graft cases the dressing develops a foul odor, especially in warm weather, even without the presence of infection, but this odor is rather different from that due to infection. The former is one of retained secretions, the latter that of suppuration, and they can be distinguished from each other. In the absence of other symptoms and signs this odor need not cause great concern, though it does mean that the dressing should be changed at the earliest time possible, consistent with proper postoperative procedure.

Cartilage repairs and bone grafts should be dressed with pressure and with a plaster of Paris or crinoline bandage, the dressing to be changed on the tenth day. A bluish discoloration of a skin graft, especially when in the central portion of a flap or in

a dermo-epidermal graft (Sheehan) is indicative of its low vitality, or even death. If this occurs that portion of the graft will undergo liquefaction followed by secondary contraction. At times, a few shallow stab wounds in this portion of the graft may reëstablish its vitality, but this discoloration is always a serious sign. Cartilage and bone grafts should be dry and the bed should remain dry to insure successful takes. Gaping of the wound, or even the appearance of serum from the wound is unsatisfactory. Infection, naturally, manifests itself by a spontaneous opening of the wound and by subsequent suppuration. It is especially important that these cases be free from hemorrhage, even capillary oozing, before they are closed.

CONCLUSIONS

The author appreciates that most of the contents herein have been published before in various texts and monographs, and also that considerable important detail has not been included. This is, however, presented as a convenient abstract of some of the forces which underlie ophthalmic plastic surgery. Textbooks on the subject must be utilized repeatedly by all in order that we may become and remain proficient in this exacting branch of ophthalmology.

A TREATISE on pathology was written in 1829 by William Edmonds Horner. This was followed by a text on pathology by Samuel D. Gross of Philadelphia, which was published in 1839, and acknowledged the best in the English language at that time.
From—"The Postmortem Examination" by Sidney Farber (Thomas).

EYE, EAR, NOSE AND THROAT SYMPOSIUM

Ear, Nose and Throat Section

EDITED BY

L. W. DEAN, Jr., M.D.

Assistant in Otolaryngology, Washington University
School of Medicine, St. Louis, Missouri

AURAL SURGERY

THEORETIC AND TECHNICAL ADVANCES IN RECENT DECADES

PHILIP D. KERRISON, M.D.

NEW YORK CITY

I WAS asked some months ago to write, as part of this Symposium, a short introductory paper on the advances of recent decades in otology, a task which I have found more difficult than at the time it seemed. Ten years out of practical working touch with any branch of medicine leave wide gaps in even such knowledge as one once possessed—gaps which it would be futile to attempt to repair.

It is probably not uncommon, as the heaping years dull the camera of the mind, that one's earliest impressions in any field of observation or study are relatively vivid and persistent; and I happened to become a student of otology in the last year of the nineteenth century. It is chiefly this fact which serves as an excuse for this inadequate report.

On the surgical side, how better could one picture the status of any branch of medicine at any particular date or period than in the routine work of a large and active hospital clinic? If therefore, we could project ourselves mentally into the busy ear clinic of the late Dr. Dench in the New York Eye and Ear Infirmary, with which I became associated in 1899, we should obtain automatically some conception of the extent of the journey we have traveled in the few brief years that have followed.

In order to present a comprehensible basis of comparison, I would like to refer briefly to such commonplace surgical procedures as myringotomies, the surgical treatment of furunculosis, the old technique of the mastoid operation (mastoidectomy) and, since it has so important a bearing on aural therapeutics, to the adenoid surgery of that time.

Myringotomies in clinic patients—adults, children and infants—were at that time performed without anesthesia. I know, or knew, of no surgical mishaps directly resulting therefrom, but have always suspected that the "myringotomies" were in many cases little more than punctures. That, I am inclined to believe, was as it should have been. It is better to provide insufficient drainage than to dislocate a stapes.

The above note is made merely to indicate a somewhat cavalier attitude toward this important little operation. Perhaps it extended to the general profession, for it was not very uncommon in the early decades to come upon reports in otologic literature of serious extension of infection due to myringotomies by general practitioners. Dr. Cunning quite recently reported a fatal case of suppurative meningitis directly due to myringotomy by the family doctor.

FURUNCULOSIS OF THE EXTERNAL AUDITORY CANAL

At the opening of the century and during the succeeding ten or fifteen years, the prescribed treatment of this exceedingly painful lesion was prompt incision, followed in clinic patients by aseptic irrigations, and in private patients by frequent changes of wet gauze dressings in the canal. Measured by results this routine method was deplorably unsatisfactory by reason of the distressing frequency of recurrences. Quickly following series of furuncles in different walls of the canal—three, four, or even as many as ten—were by no means unknown. To combat this dreary and nerve-racking series of events, vaccines, autogenous or "stock," were resorted to, but with no convincingly decisive results. With each recurrent furuncle, the routine

of treatment, beginning with incisions was repeated.

There are probably few otologists who were in active practice during the first years of the century who cannot recall cases of furunculosis of the canal which entailed more torture to the patient than that commonly experienced during the course and surgical treatment of an acute suppurative mastoiditis of average severity.

THE OPERATION OF MASTOIDECTOMY

This had been practiced, and with the saving of hundreds of lives, long before the beginning of the present century. The technique of the bone excavation in 1900 differed only in relatively minor details from that of today, subsequent developments being mainly in the direction of greater thoroughness in the removal of diseased bone. On the other hand, the treatment of the soft parts (flaps) and the postoperative treatment were faulty both in theory and execution.

The theory was that of the open wound. Tight packing of the entire bone cavity was thought to promote the formation of firm and healthy granulations. Incidentally, another advantage claimed was that it permitted the surgeon to keep the aditus under more or less constant observation. If granulations formed within the aditus, they were to be removed by careful use of a small curette. Who that was in hospital practice at that time can forget the long strips of serum or pus-soaked iodoform gauze removed at each dressing during the first postoperative weeks?

Unfortunately this method took no thought of the reconstructive value of the periosteum underlying the flaps, nor of the flaps themselves, which underwent more or less retraction and atrophy.

Repair processes may be delayed or diverted, but cannot be wholly inhibited. Too often the integument spread over the flap edges, and thence extended more or less rapidly over the walls of the bone cavity. Hence the unsightly post-auricular depressions, which were so common as sequelae of the mastoid surgery of that day.

TONSILLO-ADENOID SURGERY

The usually combined operation of tonsil and adenoid removal is so essential a factor in aural therapeutics as to demand brief discussion.

Removal of the tonsils was effected by a cleverly devised self-acting guillotome which, properly adjusted, automatically elevated the tonsil from its bed and removed it, or part of it, by a single movement. If it did not generally effect complete removal, it produced little or no avoidable traumatism, and was a safe and useful instrument, often affording much relief.

The technique of adenoidectomy during the first half-decade or more is a different story. The popular instrument at the start of the century was a cutting forceps in form somewhat resembling a rongeur. So far as I know, it was the first cutting forceps designed for this field of work, and it had the defects often inseparable from initial efforts. One cannot, however, describe the adenoid surgery of that era without reference to the defects inherent in the instrument itself.

The dangers of operating with this form of forceps were due to the disproportionate traumatism which relatively slight displacements of the instrument might cause. For example, if the blades were unduly separated in relation to the width of the nasopharynx, they might engage the walls of the fossae of Rosenmüller and injure the ears. If they were unduly approximated and inserted too far, they might enter the posterior nasal fossae, engage and bring away part of the bony nasal septum. Both of these accidents have occurred. If the surgeon were heavy-handed and exerted undue downward pressure, he might remove also the nasopharyngal mucosa, and also even some of the underlying muscle.

I think that few ear or throat surgeons who were in active hospital practice in the

first decade of this century will deny that the adenoid surgery of that period, measured by the average results, was deplorably bad. It was traumatic surgery par excellence.

Before leaving this brief consideration of aural surgery at the beginning of the century, it should be recalled that much pertinent knowledge, now at our disposal, had yet to be revealed. The physiologic reactions to irritation of the static labyrinth or vestibular mechanism were practically unknown. The symptomatology of suppurative labyrinthitis, as we now know it, had yet to be worked out, and to the rank and file of workers in otology, laybrinthine suppuration was hardly recognized as a pathologic entity. This gap in our essential knowledge may have led to a rather casual and care-free mental attitude toward tympanic surgery. In major surgery it deprived the aural surgeon of certain danger signals and may have accounted for occasional fatalities which might otherwise have been guarded against.

To epitomize: the first years of the present century seem to have been characterized by lack of surgical respect for the tympanum; by faulty and illogical treatment of auricular furunculosis; by theoretic and technical faults in mastoid surgery, and by adenoid surgery fraught with trauma.

Against this unpromising start, one may hazard the prediction that the decade from 1900 to 1910 will go down in otologic history as a period of outstanding scientific and therapeutic advance.

Surgical Progress in the Twentieth Century

Barany's Investigations of the Static Labyrinth. It was in the latter years of the first decade that the late Professor Barany, then of Vienna, published the results of his epochal investigations of the static labyrinth which, I think, reached this country in the latter part of 1907.

Barany was essentially a philosopher and physiologist rather than a practical surgeon, though he prized above all things his position as surgeon and lecturer in otology in the Vienna General Hospital on account of the enormous opportunities for work and observation it afforded. The publication of his work on labyrinthine physiology, with the description of his theretofore unknown symptom-complex of vestibular irritation, received prompt and world-wide recognition by physiologists and otologists alike.

Basing his work logically upon the older experiments of Ewald upon pigeons, Barany was able to demonstrate clearly, and therefore convincingly, that the various components of his syndrome—the nystagmus, the ataxia or falling direction, etc.—were each due to the endolymph movements in certain parts of the semicircular canal system or, as is almost invariably the case in lesions localized in one or the other labyrinth, to functional imbalance, ablation of function in the diseased labyrinth acting as practical stimulation of the sound organ of the opposite side. Furthermore, through the caloric and rotation tests, the various objective phenomena—the nystagmus, pointing deviation, ataxia or falling direction—could be studied by any intelligent student of otology with the coöperation of any normal individual or suitable clinic patient. It is not surprising that the importance of these fascinating studies captivated the imagination and attention of otologists in all parts of the world.

Unquestionably Barany's work was known in Vienna and other European centers some time before it was known in America. That suppurative labyrinthitis had engaged the attention of European olotogists long before its importance was recognized here was shown by the astonishing flood of literature dealing with surgical reports and statistics which appeared in the years immediately following. Mygind of Copenhagen recorded his belief that surgical injuries to the stapes during the radical operation was the most prolific cause of suppurative labyrinthitis. Jansen of Berlin, in 1908, reported that of 100 cases in his own surgical experience there were fifteen

fatalities directly due to surgical mishaps during the radical operation. Of these fifteen fatalities, seven were due to accidental extraction of the stapes, others to tears of the stapedial annular ligament, etc. The statistics here reported, which might easily be amplified by contemporary reports of Hinsberg, Panse and others, are old, but for precisely that reason they are important as indicating the great stimulus which Barany's work gave to the study of this greatest of all gateways of infection to the meninges.

Summary. Barany's investigations were the work, not of a professional physiologist, but of a worker and university lecturer in otology. In the flood of absolutely new light they threw upon the physiology of the static labyrinth, on the symptomatology of suppurative labyrinthitis, on the etiology and perhaps prodromal symptoms of leptomeningitis, and on the surgical danger points in temporal bone surgery, they constitute unequivocally the outstanding otologic advance of the first quarter of the present century.

THE SO-CALLED "BLOOD CLOT OPERATION" (BLAKE)

In 1906 the late Dr. Clarence J. Blake of Boston published his paper on "the value of the blood-clot" in mastoid surgery. Dr. Blake's surgical procedure contemplated no change or modification of the bone excavation, and was concerned solely with the treatment of the soft parts, or flaps, and the use of a blood clot as a reconstructive agent.

With slight modification of the initial incision, giving greater width to the anterior flap, the steps of the operation included the following:

1. Provision of adequate tympanic drainage through re-incision of the membrana tympani, when called for.

2. Great care in preserving without injury the periosteum underlying the flaps.

3. At conclusion of the bone operation, thorough cleansing of the mastoid excavation with sterile normal salt solution.

4. The bone cavity, having been dried with sterile gauze, was then allowed to fill with blood. The flaps were then brought into careful coaptation and held by interrupted sutures of silkworm gut, closing the entire wound except at the lower end, which was left unsutured for the escape of serous discharge.

The theory of the blood-clot was based mainly on two hypotheses: (1) that the blood—as likewise the clot—possessed an inherent bactericidal value which might be counted upon to combat or control wound reinfection; and (2) that, by analogy with known bone-forming value of the clot in certain destructive lesions of the long bones, it should have like reconstruction value in mastoid surgery.

Following the publication of Dr. Blake's paper, others appeared, notably those of the late Drs. H. O. Reik and F. B. Sprague, discussing the value of the blood-clot theory. More decisive, perhaps, were the numerous case histories published by aural surgeons in various parts of the country, the results reported varying from almost "first intention" healing to breaking down of the wound from reinfection. Even in the latter cases, however, it was not difficult to reopen, resterilize and resuture the wound, often with great shortening of the postoperative period.

Viewed in retrospect, it seems unfortunate that the term, "blood-clot operation," was ever applied to Dr. Blake's very useful modification of the old technique; for the blood-clot theory, so far as I know, has long since been abandoned. Had he based his method squarely on the reconstructive value of the preserved periosteum and of the better local blood supply his theory and results would have gone hand in hand and left no room for argument. As it is, he taught aural surgeons the great value of the closed or partially closed wound in mastoid surgery.

From the viewpoint of humanitarian and practical results, i.e., the saving of pain and discomfort of hundreds of patients, and considerable average reduction of the post-

operative period and the practical elimination of post-auricular scars and depressions, Dr. Blake's modification seems to me to deserve a place among the notable practical advances of the early years of the century.

The second and third decades seem to have been a necessary period of consolidation, i.e., of unspectacular, but still exceedingly important technical progress. The lessons of Barany's work familiarized surgeons with danger signals hitherto unknown or not sufficiently recognized, which impelled greater surgical care. Jansen's complacent and naive report of fifteen fatalities in his own radical operations as directly attributable to surgical mishaps (stapedial extructions and luxations, etc.) supplied a striking, though hardly needed lesson in what otologic surgery should not be. In short the second and third decades constituted a period of practical technical progress based on the ideal of reduced mortality and the reduction of human suffering.

TONSIL AND ADENOID SURGERY

In no phase of otolaryngologic surgery has the technical advance been more definite and humanly valuable than in the surgical removal of tonsils and adenoids. It has been a gradual evolution in which mechanical ingenuity in the invention of new instruments and the development of new techniques have gone hand in hand.

If, under the impetus of recognized surgical aims and needs, tonsil surgery has improved, the changes in the technique of adenoidectomy have been far greater. Here even more than in the case of tonsillectomy, the advance has been marked by a succession of new instruments—instruments involving changes of mechanics or the method or direction of applied force.

With regard to adenoid surgery, at least, it may be said (a) that with the instruments in use at the start of the century, the clean surgical results of today would have been practically impossible; and per contra (b) that with modern instruments, e.g., the La Force adenotome, it would be difficult, if not impossible, to produce the lacerations which were not uncommon in the first decade.

FURUNCULOSIS OF THE EXTERNAL AUDITORY CANAL

The treatment of this exceedingly painful lesion, with its usually unhappy results, has already been described.

As we now know, the theory of incision to evacuate confined pus, though the logical procedure in most other parts of the body, fails as logically in furuncles of the auditory canal by reason of anatomic conditions peculiar to that region. Here we deal with an encapsulated collection of pus in a relatively rigid cartilagino—osseous canal, but surrounded by an area of intense surface inflammation. By incision we obey the surgical law of providing exit to confined pus, but simultaneously we open the gateways to wide pus diffusion through surrounding vascular engorgement to all parts of the canal.

Somewhere, I think, about the middle of the second decade, the routine incision of furuncles of the canal was abandoned by the best surgeons, the substitute treatment being by fairly firm packing of the canal with sterile wet gauze wicks. This change was followed—in my experience at least—by practical cessation of recurrences.

In the important matter of minimizing human suffering, this change of theory and practice deserves inclusion among the real therapeutic advances of the period. It was long overdue.*

SURGERY OF THE FACIAL NERVE

Unquestionably one of the outstanding events in otologic surgery in the present century has been the brilliant culmination of the long years of patient and difficult research, first upon animals and then tentatively upon man, which finally removed destructive surgical accidents to

* N.B. As qualifying what has been said, it is of course recognized that there are neglected cases in which, both to relieve infection and to protect the integrity of the canal, the knife becomes the only recourse.

the facial nerve from the list of incurable lesions.

Recognizing, as of course we all do, two stages in the evolution of facial nerve surgery, I think the two phases should be separated in our minds as two separate and distinct surgical efforts, i.e., the *first* dealing with anastomosis of the peripheral segment of the facial with another motor nerve; and the *second*, far more difficult by reason of the multiplicity of problems involved, which restores to the injured facial its inherent and normal function.

Probably a full list of the printed records, papers and discussions of the late Sir Charles Balance on this subject would very nearly cover all that need be known of the earlier chapter on the surgery of the facial nerve. The history of Sir Charles' experiments may be sufficiently indicated by the simple enumeration of the nerves selected, given in the chronological order in which they were utilized and later discarded. They were (1) the spinal accessory; (2) the hypoglossal; and (3) the glossopharyngeal. Apparently each or any of these nerves, if anastomosis with the distal segment of the facial was successfully accomplished, yielded satisfactory results so far as preventing atrophy of the facial muscles; but full restoration of normal, subconscious, automatic facial movement was rarely, apparently never, attained.

After sufficient trial, the spinal accessory was discarded because, to use Sir Charles' words, "the main result of facial-spinal accessory anastomosis, and often the only result, was associated movements of the face and shoulder." He cites one case, in the practice of another surgeon, in which facial movements could be initiated only by voluntary movement (raising) of the homolateral arm.

The second nerve utilized, the hypoglossal, gave hardly more satisfactory results. In a later paper Sir Charles reported that facio-hypoglossal anastomosis too often resulted in uncontrollable and often grotesque facial movements, perhaps more distressing than the paralysis and disfigurement the operation was designed to remove. He therefore decided to abandon both the spinal accessory and the hypoglossal, and experiment with the glossopharyngeal.

Perhaps nerve anastomosis as applied to a divided facial failed of favorable results because it outrages some law of nature. Such a hypothetic law may perhaps be stated as follows: various (perhaps all) motor nerves may supply *potential* motor power to the peripheral segment of any divided motor nerve; *but to expect them to supply such extraneous motivation without simultaneous excitation of the muscle groups which their central nuclei were designed solely to motivate or control, is to count upon the abnormal, and therefore upon the improbable.*

SURGICAL REPAIR OF THE FACIAL NERVE

It was the most fortunate of fortuitous events that brought the late Sir Charles Balance and our beloved friend, the late Dr. Arthur B. Duel, into effective collaboration. It would be impossible to imagine a man more uniquely qualified, both technically and by innate qualities of mind, character and ability to plan, coördinate and work so effectively in bringing this great undertaking to a successful issue. Aside from the composite technical difficulties, the practical and material problems and obstacles—the obtaining of animals for experimentation, of suitable operating and housing quarters for animals under surgical care, of proper food supplies, of necessary attendants and helpers, and the difficult problem of providing the essential financial support—probably fell solely or chiefly on Dr. Duel.

In my early years in New York I saw much of Duel, and he possessed then certain qualities, illusive or difficult of definition, which gave to his personality a certain charm or glamour. This, to me, he never seemed to lose.

It is always interesting, in the case of men of exceptional force and ability, to ponder over the qualities, moral, mental or

of whatever nature, which enable them to achieve their results. I think difficulty appealed to Dr. Duel rather than deterred him or inhibited his powers. Also he undoubtedly possessed an innate force which in his contacts with men claimed their immediate confidence, first in himself as a man, in his absolute sincerity, and therefore in the projects he espoused and his ability to carry them to a successful issue. On no other hypotheses can be explained his ability in a time of nation-wide depression and financial stress to obtain the financial support essential for so costly an enterprise.

It would be useless, even were I competent to do so, to attempt a full and adequate description of the researches which are so fresh in otologic memory. The experiments, which were continued over two years' time, were conducted at Dr. Duel's country home. The animals used in the experiments included monkeys, cats, rats and possibly others. During the first year Sir Charles and Dr. Duel worked in collaboration; during the second Dr. Duel worked alone.

The purpose of the research was to investigate the possibilities and technique of "direct line repair" of the divided or injured facial by means of an autoplastic nerve graft. The nerve used in the later experiments and the successful operations on patients was the femoral cutaneous—probably the anterior; but it was definitely demonstrated that either a motor or sensory nerve might be used successfully. Duel stated that facial nerve gaps of any length can be bridged by such autoplastic grafts.

During the second year of this research, when Duel worked alone, he proved that more rapid and, on the whole, better results were obtained if the nerve from which the graft was to be taken had already undergone Wallerian degeneration. This preliminary work was effected by incising the nerve to be utilized and allowing it to remain in situ for a period ranging from two to three weeks by which time, Wallerian degeneration is said to be advanced or complete.

The results of this logical surgical method we all know—subconscious, automatic, emotional response, as compared with associated movements, often grimacial in effect, frequently resulting from anastomosis between two functionally alien nerves.

What wonder that Duel's fame spread rapidly beyond our national boundaries, and that he was called to Europe to demonstrate before various otologic bodies the difficult surgical technique for which his genius was so largely responsible. Probably the death of few men of our time has caused so profound a sense of sadness and personal loss to friends and confrères both here and abroad. But to the man himself—to any man—what better time to die than at the conclusion of a great achievement?

OTITIC CEREBROSPINAL MENINGITIS

Are we nearing the end of the long battle against that scourge of humanity, suppurative cerebrospinal meningitis?

The woeful past history of surgical intervention may almost be told by bare mention of the methods employed: (1) repeated lumbar punctures, to relieve pressure; (2) multiple dural punctures; (3) Haynes' operation of drainage of the cisterna magna; (4) drainage of the lateral ventricle; (5) through-and-through cerebrospinal lavage. All of these methods were tried out twenty years ago by Dr. E. W. Day, then of Pittsburgh, and other surgeons. In the past two decades various modifications—all based on one or other of the five surgical measures listed above—have been hailed as the sine qua non only to fall by the surgical wayside. Dr. Eagleton of Newark has given much time and study to the problem of surgical treatment and his papers and discussions have doubtless been of value in keeping the problem under constant discussion and study. But until the discovery of sulfanilamide and its compounds, no surgical method was known which could be relied upon to produce an

appreciable dent in the frightful average mortality of scientifically diagnosed cases of suppurative cerebrospinal leptomeningitis.

Within the scope of this condensed report the most important and striking fact is the almost fantastic difference between the old (i.e., pre-prontylin) statistics of mortality, and those of recent reports.

Dr. Dwyer's review of collected statistics of mortality results places the recoveries as less than 4 per cent. The following pre-prontylin statistics were taken from Dr. Cunning's not yet published paper:

	Per Cent
Dr. Josephine B. Neal, of the New York Department of Health, of 258 cases, recoveries.....	5
A. A. Gray, 2,200 cases (1900–1935).....	3
Manhattan Eye, Ear and Throat Hospital.....	2
Johns Hopkins Hospital, no recoveries.	

Combining the above, I think we may assume that 3 per cent quite, or more than quite, covers the old average recovery percentage under surgical treatment. Dr. Neal's series of 258 scientifically diagnosed cases gave the highest recovery percentage reported.

Against the above we may place two small but tremendously impressive series in which sulfanilamide has been used:

Dr. Neal: 11 cases: 2 deaths and 9 recoveries
Dr. Cunning: 9 cases: 3 deaths and 6 recoveries.

Combining the two series of eleven and nine cases respectively, we have the following result: 20 cases with 5 deaths; recovery rate—75 per cent.

In all of Dr. Neal's cases, the germ was the Streptococcus hemolyticus. Of Dr. Cunning's series the following facts may be noted: One case, pneumococcus, type III, was apparently of the acute or fulminating type, the patient dying on the day following admission to hospital. In the other two fatal cases in Dr. Cunning's series, the infection showed the same germ, pneumococcus, type I. In one of the latter fatal cases, infection was obviously the direct result of myringotomy by a general practitioner—probably a case of stapedial extraction or tear of the stapedial annular ligament, as indicated by immediate supervention of acute vestibular symptoms (nausea, vertigo, nystagmus, etc.) and systemic signs of sudden and overwhelming sepsis. Six cases which recovered all showed by culture Streptococcus hemolyticus, as did all Dr. Neal's cases.

Dr. Cunning's description of surgical details is interesting as we see some of the oldest of hoary-headed surgical measures revived, modified and dovetailed into a surgical systematic routine. The "multiple dural punctures" of the ancient fathers are replaced by superficial linear slits of the dura in parallel lines over the region to be drained—surely a safer and more logical provision for maintained drainage and relief of pressure. The elevation of the foot of the bed and suturing of the auricle forward to the face are logical adjuvants to the above.

The two agencies depended upon to save the patient are: (a) proper administration of sulfanilamide, and (b) frequent small blood transfusions.

Doctors Neal, Cunning and Dwyer all state and reiterate that sulfanilamide or its compounds should be used intensively, i.e., to whatever may be the safe or physiologic limit.

For surgeons generally—for otitic meningitis cases are of necessity surgical cases—Dr. Dwyer, considering dosage as based on an adult body-weight of 140 pounds, endeavors to fix approximately a safe daily amount of sulfanilamide according to the severity of the lesion as follows: mild case, 30 gr. (6 tablets of 5 gr.); medium case, 60 gr.; severe case, 120 gr. daily.

In qualifying this, he makes the following philosophic comment: "There is no standard dose . . . medicine deals with human beings, not machines, and cannot be practiced by rule of thumb. . . . There is no help for it but to realize that the correct dose of any drug is that which will produce the desired effect." This modification seems to me the correct scientific attitude toward a new and powerful drug, for it places the individual responsibility upon the phy-

sician to safeguard his patient by obtaining the best available advice where his knowledge fails.

The symptoms characteristic of the drug itself are cyanosis, dizziness, and headache, which in moderate degree are considered normal reactions and not alarming. The most serious drug reactions are excessive acidosis and, of course more dangerous, hemolytic anemia. Therefore, daily urinalysis, and a blood count and hemoglobin test on alternate days are essential.

In favorable cases under prontylin medication, frequent examinations of the spinal fluid are important to determine the persistence, diminution or disappearance of the germ. The microorganism may disappear wholly from the spinal fluid and then, after several days of complete absence, may suddenly reappear, calling for immediate increase of specific dosage.

The deductions from the above are clear: with the clearing up of the clinical picture and disappearance of the microorganisms from the spinal fluid, the dosage may be reduced, *but not discontinued.* Small doses of the drug and periodic examination of the spinal fluid *over a period of at least a month after disappearance of the germ* are considered essential to safety.

SUMMARY OF TWENTIETH CENTURY PROGRESS

On the surgical side, what may be shown to the credit of otology and allied branches of medicine during the first thirty-eight years of the twentieth century?

1. Furunculosis of the external auditory canal, once the most excruciating of aural lesions, now yields readily to logical treatment, with recurrences reduced to a minimum.

2. Adenoidectomy, once the crudest and cruelest of surgical operations, now is a technically clean surgical procedure, practically devoid of trauma.

3. Technical modifications of the operation of mastoidectomy have reduced the average postoperative period by about one-half, and with elimination of post-auricular scars and depressions.

4. Barany's researches in the physiology of the labyrinth have broadened enormously the scope of scientific otology, with particular benefit to temporal bone surgery.

5. The symptom-complex of vestibular irritation has supplied surgical danger signals theretofore beyond our ken. All temporal bone surgery is far safer than at the beginning of the century.

6. The Balance-Duel operation of direct-line repair of a divided facial nerve has brought the first promise of real relief to a fortunately limited class of dire unfortunates.

7. Otitic suppurative cerebrospinal meningitis is apparently removed from the class of relatively incurable diseases.

I wish to express my thanks to Dr. Dwyer and Dr. Cunning for having given me the privilege of reading in manuscript their exceedingly interesting and instructive papers, not yet in print.

ACUTE ENCEPHALITIS OR TOXIC ENCEPHALOPATHY SIMULATING BRAIN ABSCESS*

PAUL J. ZENTAY, M.D.

Instructor in Clinical Pediatrics, Washington University School of Medicine

ST. LOUIS, MISSOURI

THE knowledge that acute intracranial processes of infectious or toxic nature may occasionally produce clinical pictures very closely resembling brain abscess is by no means new. Quite a few articles are available on this subject in the literature of the past twenty or thirty years. It is not my purpose at this time to give a review of these publications, and only a few necessary references will be made to them. The otologic journals show consistently the liveliest interest in the problem. This is hardly surprising in view of the fact that brain abscess is of great practical importance to the otologist. It represents a borderline problem for the otologist, the neurologist and the neurosurgeon.

Recognition of such an imitative process, diagnosis of a so-called "pseudo-brain abscess" is more than of passing significance. Treatment and prognosis are so vastly different in the two conditions that the welfare of the patient is largely dependent on our ability to differentiate between them.

Almost any general or children's hospital has on its files a few records of patients in whom a diagnosis of brain abscess was made on apparently good evidence, only to be proved erroneous by subsequent developments. Many of these patients with "brain abscess" improved rapidly or recovered completely while under observation. Others were promptly operated on by competent neurosurgeons, but no suppurative process was found in the region to which topographical diagnosis so clearly seemed to point. Some of these patients recovered after craniotomy, while others succumbed and autopsy proved that death was due to some intracranial process other than abscess. A few patients died without operation, but only some form of an acute diffuse process without any suppuration was found on post-mortem examination.

As already evident, any intracranial process capable of leading to such development must be without doubt a very successful imitator. The process usually begins more or less suddenly as a secondary accompaniment or a manifest complication of an underlying infection. The greatest difficulty will arise, of course, if the primary infection happens to be suppurative otitis media with mastoiditis or with sinus thrombosis.

The onset which marks the invasion of the central nervous system is usually characterized by fever, possibly by leucocytosis, malaise, and loss of appetite. In the further course the fever may continue high, and headache, vomiting, disturbance of consciousness may follow. Signs of intracranial pressure develop and papilledema may be present in a good many cases. Often focal signs develop and they add considerably to the confusion. These focal neurologic signs vary greatly and point either to the temporal lobe or to the cerebellum, or to other parts of the brain. The cerebrospinal fluid may show changes identical with those found in brain abscess.

If the intracranial process is of a very acute nature the patient may succumb within a short time. In such a case the focal signs are gradually overshadowed by the picture of a more or less general involvement of the nervous system before the patient dies. In the milder forms there is

* From the Department of Pediatrics, Washington University Medical School, St. Louis Missouri.

considerable improvement within a few days and in the great majority of the cases there is complete recovery with disappearance of the general and focal signs.

Such is briefly the composite clinical picture of these processes. In individual cases, of course, this picture may vary greatly and in practical experience hardly any case will fit into the above description exactly. This is hardly to be wondered at if we consider the great variety of intracranial processes that may lead up to such developments. The causes are manifold and the picture is truly protean. Pitfalls are abundant and errors in diagnosis are extremely easy.

As an attempt to establish some kind of order in this chaos let us consider the possible underlying processes:

1. *"Pseudo-Brain Abscess" or Localized Non Suppurative Encephalitis.* Adson,[1] Symonds,[2] Romantzeff,[3] and more recently Nielsen and Courville,[4] described a clinical picture complicating otitis media that they called "pseudo-abscess of the brain." The picture is that of a brain abscess as a rule in the temporal lobe, rarely in the cerebellum. It does not develop beyond the initial stage, no suppuration is found and the patient recovers. According to these authors the underlying process is produced by an extension of the infection along the lymph spaces exactly in the same manner as in true abscess. The pathologic process, on the other hand, does not develop beyond the stage of a localized non-suppurative encephalitis. If this theory is right, the correct name for the process should be "abortive brain abscess" rather than "pseudo-abscess." The experimental and pathologic proof for the correctness of their supposition is still lacking. On theoretical grounds there is no reason to doubt that with good tissue resistance and not very highly virulent infection such abortion of a brain abscess is a possibility. In any case the clinical picture is an established fact confirmed by many observers.

2. *Acute Toxic Encephalopathy.* This condition frequently complicates severe infectious diseases. It seems, on the basis of our present knowledge, that it is caused by bacterial toxins. These poisons apparently affect the central nervous system and produce intense edema, swelling and severe congestion, and possibly disintegration of the ganglion cells. The clinical picture is characterized by high fever, vomiting, convulsions, disturbance of consciousness, and often by general muscular rigidity. The cerebrospinal fluid is under high pressure and may show an increase of globulin. The optic discs are often congested or edematous.

If such a process appears in connection with serious infectious diseases like scarlet fever, pneumonia already complicated by otitis media and mastoiditis, it may very easily stimulate brain abscess.

The usual course is brief and recovery follows, but in the more severe forms permanent damage may result or the outcome may be fatal.

3. *Acute Disseminated Encephalomyelitis.* Nielsen and Courville in their classification of cases of "pseudo-abscess of the brain" describe a syndrome with convulsions or paralysis. They also report a patient in whom otitis media and mastoiditis were followed by loss of consciousness, general and focal convulsions, left hemiplegia, choked discs and residual hemiparesis; exploration was negative. For the explanation of the process they assume an extension of the infection from the middle ear by way of the superficial veins of the cortex toward the parietal lobe.

Evidence of course is lacking that an extension in that manner is ever possible. The clinical picture on the other hand fits our concept of disseminated encephalomyelitis perfectly.

Encephalomyelitis does occur as a para- or post-infectious process with almost any infection, and most commonly with measles. Its vagaries are almost inexhaustible. The general and focal manifestations produce the most varied pictures. In the majority of the patients the condition is transitory and complete recovery follows.

Often permanent damage results in the form of paralyses, deterioriation or loss of functions.

If disseminated encephalomyelitis develops in a patient with middle ear infection, the clinical-neurologic picture may easily simulate brain abscess for a short period. This is true particularly in those cases where the focal signs point to the temporal lobe or to the cerebellum. The clinical findings inclusive of choked discs and the changes in the spinal fluid may also be rather confusing.

4. *Epidemic Encephalitis.* Type A encephalitis has been rather rare in recent years. It may occasionally simulate brain abscess particularly if it appears in the so-called pseudo-tumoral form accompanied with choked discs.

Type B encephalitis in the St. Louis area may appear in its early stages in a form that suggests brain abscess.

Both of these conditions of course will cause embarassment only if by coincidence they occur in an individual who is suffering with acute or chronic suppurative middle ear infection or sinus infection.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Acute intracranial processes of the types discussed in the preceding paragraphs at times do simulate brain abscess very successfully. They lead to serious confusion occasionally, but will mislead the careful observer only for a short time. They are capable after all of simulating only a brief phase in the course of development of brain abscess, i.e., the stage of invasion or the stage of meningeal irritation at the height of an abscess. The rest of the clinical course of these conditions and that of brain abscess is so vastly different that separation should not be difficult.

The fact to bear in mind is that brain abscess is a localized bacterial encephalitis accompanied often by diffuse intracranial symptoms and signs. The other simulating conditions with the exception of "abortive abscess," are on the other hand, diffuse acute intracranial processes accompanied by focal manifestations which may dominate the clinical picture for a period. This is the chief reason for the confusion encountered in diagnosis. Differentiation is possible on the basis of characteristic features in further developments. In acute encephalitis or toxic encephalopathy maximum damage is invariably done at the onset. After this initial phase there is improvement and gradual return, at least, of the residual functions. In brain abscess, on the contrary, after the first stormy stage of invasion there is a more or less slow but steadily progressive development. The characteristic features of the later stages are largely identical with those of a brain tumor growing in the region where the abscess happens to be.

Therefore, careful history of the initial symptoms is essential for diagnosis. Exact observations on the specific nature of the acute intracranial process, particularly of the disturbance of consciousness, of convulsions with careful regard as to their distribution, early changes in muscle tonus and appearance of paralyses—these all will help greatly in arriving at a correct diagnosis.

Unfortunately clinical laboratory examinations are of very limited value. Changes in the leucocyte count or in the spinal fluid are not characteristic for any of the conditions.

Last, but not least, it is important to remember that there is practically never an emergency situation to face. Expectant observation hardly ever will do any harm in these conditions. On the other hand, it is more than likely to bring a correct solution of the dilemma within twenty-four or forty-eight hours.

Important immediate decision is necessary only in the case of the so-called abortive brain abscess. Here the question is whether a true abscess is developing or not. As stated before, we are dealing with an extension of the infection that may be arrested as a localized encephalitis. If the symptoms quiet down and the patient

continues feeling well the answer to the problem is evident.

Waiting and observation must be the procedure of choice wherever there is any doubt of an acute process simulating brain abscess, or if there is the least suspicion of such developments.

In toxic encephalopathy the decision may not always be easy to make in the first twenty-four hours, but the very acute onset with early evidence of increased intracranial pressure and the rather violent diffuse symptoms should help in making a correct differential diagnosis. Again observation for twenty-four to forty-eight hours and delaying of operative procedures will be more beneficial than harmful.

The question that always arises with all these conditions is, of course, shall a craniotomy be performed or not. I cannot see that there is any reason here for haste. In these patients, almost without exception, we are facing an acute process in its inception and even though the possibility of an abscess has to be considered, there is no emergency and no immediate operation will be needed. On the other hand, in the great majority of the cases a delay of a few hours will make an operation unnecessary and thus will minimize the risks.

It is stated, of course, that craniotomy and exploration are harmless measures. Even if this is conceded, it does not seem likely that a neurosurgeon would be anxious to operate on these patients if in any manner it would be possible to arrive at a correct diagnosis, or if the probability of an abscess could be greatly shaken by continued careful observations.

REFERENCES

1. Adson, A. W. Pseudobrain abscess. *S. Clin. North America*, 4: 503, 1924.
2. Symonds, O. P. Some points in the diagnosis and localization of brain abscess. *J. Laryng. & Otol.*, 42: 440, 1927.
3. Nielsen, J. M., and Courville, C. B. Pseudo-abscess of the brain, etc. *Ann. Otol.*, 43: 972, 1934.
4. Alderson. Suspected brain abscess. *Ann. Otol.*, 19: 755, 1910.
5. Jack. Symptomatology, diagnosis and treatment of encephalitis and brain abscess. *Ann. Otol.*, 14: 722, 1905.
6. Langsteiner, F. Encephalomyelitis and Stauungspapille. *Münchcn. med. Wchnschr.*, 84: 406, 1937.
7. Hutcheson, J. K. Encephalitis simulating brain abscess: case report. *Kentucky M. J.*, 26: 300, 1938.

SINUS THROMBOSIS

I. FRIESNER, M.D.,
Consultant Otologist, Mount Sinai Hospital

J. G. DRUSS, M.D.,
Adjunct Otologist and Assistant Otopathologist, Mount Sinai Hospital

J. L. GOLDMAN, M.D.
Adjunct Laryngologist, Mount Sinai Hospital

AND

H. ROSENWASSER, M.D.
Adjunct Otologist, Mount Sinai Hospital

NEW YORK CITY

INTRODUCTION

THE care of the patient ill with otitic sepsis presents problems of greatest interest. If one considers that in sinus thrombosis there is not only the possibility of complication by direct extension but also through the microorganisms circulating in the blood stream and that no body tissue is immune from metastatic lesions, it can readily be grasped how widespread the clinical interest must be.

In discussing general invasions of otitic origin, Libman[1] emphasized the necessity for employing precise terminology lest one draw conclusions which may result in harm to the patient. Therefore for purposes of clarity we repeat previously made explanations of those terms whose meaning must be precise and clear. First, by a general invasion we mean the entrance of bacteria into the general circulation. According to Schwartzman the term "invasion" can be subdivided into (1) transitory bacteremia, and (2) septicemia. While the clinical concept expressed by the term septicemia might have been clear, it has unfortunately been loosely used, and as a result its meaning is now so indefinite that it had better be eliminated and the term bacteremia substituted. If one wishes, the terms streptococcemia, staphylococcemia or pneumococcemia, whose meaning is unmistakably clear, may be used interchangeably with bacteremia. A general invasion then, is a bacteremia, and it may be transitory or persistent. The criteria for this differentiation have been completely described by one of us (I. F.[2]) in a previous communication and need not be repeated here. The persistent bacteremias resulting from lateral sinus thrombosis or phlebitis have been of utmost significance diagnostically, prognostically and as an indication for therapy.

BACTERIOLOGY

It seems fair to state that of all phlebitic infections, lateral sinus thrombosis presents the most constant features. In no other disease is one particular venous channel so regularly and consistently involved. For this reason the bacteremia which is always associated with lateral sinus thrombosis becomes the most important clinical finding of the disease. The recognition of the bacteremia is of paramount importance in the diagnosis of the condition, and is of value in interpreting course and prognosis. In view of this the significance of interpretation of blood cultures in lateral sinus thrombosis deserves special consideration.

Of the great contributions to otology, Libman's original work[1] on lateral sinus thrombosis occupies an eminent position. Further investigations and studies on the subject have corroborated his observation that a bacteremia can be demonstrated very often in this disease and that the invading microorganism is usually the Streptococcus hemolyticus (Beta-Brown).

The infected thrombus, which may either be attached to the wall or occlude the lumen, constantly releases thrombotic particles infected with microorganisms into the blood stream. This is responsible for the characteristic clinical picture of chills and septic fever. Thus it is reasonable to conclude that lateral sinus thrombosis is always associated with a bacteremia at

some time during the course of the disease. This is especially true in the early stages of the formation of the thrombus. Inasmuch as this bacteremia is the only uncontradictable direct evidence that this condition offers, the diagnostic importance of blood culture findings becomes obvious.

The microorganism isolated from the infected blood stream in lateral sinus thrombosis is usually the Streptococcus hemolyticus and very rarely the pneumococcus type III. It appears almost as if this type of phlebitis is essentially a disease of Streptococcus hemolyticus origin. Although the hemolytic streptococcus is also the predominant etiologic organism in acute mastoiditis, the Streptococcus viridans and non-hemolyticus, the Staphylococcus aureus and albus and various types of pneumococci have been isolated from infected mastoid bones. It would appear from these considerations that the Streptococcus hemolyticus and pneumococcus type III possess in certain instances of acute mastoiditis a predilection to invade the lateral sinus. The virulence and power of invasion of these microorganisms and possibly their selective affinity may be the responsible factors in predisposing the venous sinus to infection.

Rarely such a microorganism as B. proteus and B. pyocyaneus may become pathogenic and lead to infection of the mastoid bone and also the lateral sinus. When this occurs, the nature of the infection is usually an acute exacerbation of a chronic middle ear suppuration. In our experience, infection with each of these microorganisms resulted in a fatal issue and happened to have occurred in children.

The success with which bacteremias are demonstrated is largely dependent on the blood culture methods employed. Inasmuch as the number of microorganisms which circulate in the blood stream is very often scant and as there is a natural mortality among microorganisms, certain principles for the maintenance and enhancement of bacterial growth should be observed to obtain good blood culture results. In any blood culture technique the features necessary to obtain a high percentage of positive blood cultures are the inoculation of a large quantity of blood, the use of various and rich mediums, daily spreads, subcultures and prolonged observation. The blood culture technique employed at the Mount Sinai Hospital is described in complete detail elsewhere.[3]

Occasionally a strictly anaerobic Streptococcus hemolyticus is responsible for a lateral sinus phlebitis. It is necessary, of course, to use suitable anaerobic mediums to recover and grow out this microorganism from drawn blood. At the present time an anaerobic medium is employed routinely in the blood cultures at our institution.

In the consideration of the interpretation of the blood cultures, much attention should be paid to the fluid mediums. Very often the microorganisms, and we refer especially to the Streptococcus hemolyticus, grow out only in the fluid mediums and not on solid mediums. To obtain growth in a suitable fluid medium, inoculation with only a very small number of microorganisms may be necessary. They will multiply until the maximum concentration for the particular medium is reached. On a solid medium, one microorganism will yield only one colony. When it is realized that the mortality of bacteria on solid medium is considerably greater than in fluid medium, the value of the fluid medium in culturing microorganisms can be readily appreciated. In a large series of cases of lateral sinus thrombosis which we have reviewed,[4] growth was obtained in the fluid mediums alone in 50 per cent of the preoperative blood cultures. The solid mediums are of value largely in the quantitative determination of the concentration of microorganisms in the blood stream.

Even among the various fluid mediums, it is not an infrequent occurrence to obtain growth only in the most luxuriant medium. The finding of the Streptococcus hemolyticus in one flask of medium has the same import as discovering growth in all the mediums. The establishment of the diag-

nosis and the decision to operate remain the same.

This brings us to the consideration of the fluid mediums only, even on one occasion in a single flask, is suggestive of a sinus wall infection, if all other clinical possibilities

Fig. 1. Thrombophlebitis of the sigmoid sinus. Note the inflammatory reactive changes in the adjacent bone marrow of the petrous pyramid. ss, sigmoid sinus. b, bone marrow.

number of microorganisms found on blood culture and accordingly the number present in the blood stream. When the microorganisms circulating in the blood stream are few in number, they are more difficult to grow out on blood culture. Thus the growth in fluid mediums alone, especially in one fluid medium, and not on solid mediums indicates a paucity of microorganisms in the circulation. From a purely diagnostic point of view, however, the numbers of microorganisms present in the blood stream in lateral sinus thrombosis are of little significance. When dealing with the Streptococcus hemolyticus, the presence of even a few microorganisms in the blood stream, or the growth of the microorganism in only one flask, indicates definitely the existence of a source of infection. Thus, in a suspected case of lateral sinus thrombosis, growth of Streptococcus hemolyticus in

have been carefully considered and excluded. Accordingly, such a finding under these circumstances is also an indication for operative interference. The same view may be entertained for pneumococcus type III, but for no other microorganism in this condition. Before any other bacterium is regarded as the causitive agent in a suspected or doubtful case of lateral sinus thrombosis, it should be isolated from both solid and fluid mediums on one occasion, or from fluid mediums on two occasions.

Since these facts concerning lateral sinus thrombosis have been fully appreciated, blood culture studies have been especially helpful in solving septicemias with vague otitic histories and inconclusive physical findings. The discovery of the Streptococcus hemolyticus in such cases has led to the diagnosis of lateral sinus thrombosis with consistent accuracy, after all other

sources of infection have been excluded. Conspicuously illustrative of such instances is the type of sinus thrombosis known as primary jugular bulb thrombosis.[5] These patients, when examined, usually present a healed or healing middle ear infection and no evidence of mastoiditis. The history of a recent otitic infection and the presence of a bacteremia suggest the possibility of a primary jugular bulb thrombosis, but the finding of a hemolytic streptococcus blood stream infection establishes the diagnosis when no other focus of infection can be ascertained clinically.

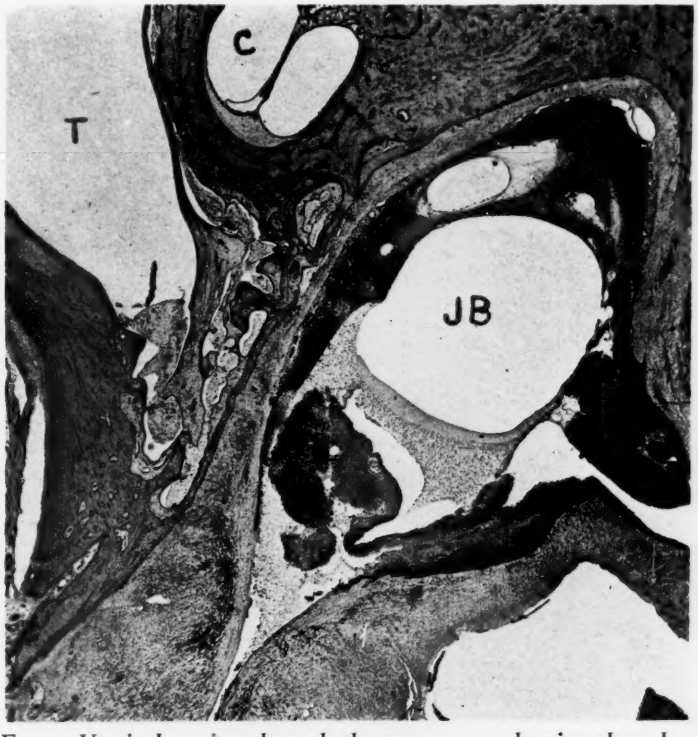


FIG. 2. Vertical section through the tympanum showing thrombophlebitis of the jugular bulb. Free purulent exudate is present within the bulb. There were multiple dehiscences in the jugular dome. C, cochlea. JB, jugular bulb. T, tympanum.

Repeated sterile cultures, we also believe, represent information which should be seriously considered in the evaluation of cases under observation. An otologist who has reliable bacteriologic studies at his disposal should be disinclined to operate in a suspected case of sinus thrombosis with repeated sterile blood cultures, unless undeniable clinical findings are encountered. This policy has borne satisfactory results, for such patients after a period of observation have manifested indications of pneumonia, pharyngeal infection, pyelonephritis, meningitis, or some other illness which accounted for the clinical course.

Blood cultures are also very helpful in the postoperative management of lateral sinus thrombosis. Repeated growth of the microorganism in cultures after operation suggests that the infected venous channel has not been entirely obliterated or blocked off. This persistence of a bacteremia usually indicates that further operative interference is required. It may be necessary to obliterate more of the torcular end of the lateral sinus or to evacuate an abscess in the jugular bulb. Of course, at times when the petrosal sinuses and other channels are involved, the phlebitis is beyond surgical approach.

Blood cultures can play an important rôle in the differential diagnosis of conditions which present the clinical picture of sepsis in association with otitic infections.

First of all, we wish to state that in our experience infections of the middle ear and mastoid bone are not associated with genetic Streptococcus hemolytic bacteremias undoubtedly have their origin in pharyngeal and nasopharyngeal infections.

FIG. 3. Thrombophlebitis of the superior petrosal sinus. Suppurative process in the adjacent bone and in the antrum can be seen. A, antrum. SPS, superior petrosal sinus.

bacteremias. An otitic infection with a positive blood culture, especially of Streptococcus hemolyticus, strongly suggests the existence of a lateral sinus thrombosis or primary jugular bulb thrombosis.

The complication which is very liable to confuse the clinical picture of an otitic infection is erysipelas. Erysipelas in the earlier stages of the disease may produce a bacteremia, and the microorganism isolated from the blood stream is always the Streptococcus hemolyticus. The confusing feature of the disease is the fact that the bacteremia may exist during the first day or two of the illness before the erythematous lesion appears. This observation has been particularly noted in children. Pharyngeal and nasopharyngeal infections may also produce Streptococcus hemolyticus bacteremias, but the portal of entry in these instances may be difficult or impossible to uncover. Many of the so-called cryptogenetic Streptococcus hemolyticus bacteremias resulting from meningeal and pneumonic infections are as a rule readily differentiated and diagnosed by evident manifestations and findings relative to the particular organ involved.

As already stated, it appears that only the type III pneumococcus of all the pneumococci produces a phlebitis of the lateral sinus. This occurrence is also comparatively rare. Thus, blood culture studies are of inestimable value in the differential diagnosis of pneumonic infections associated with otitic infections. The finding of a pneumococcus bacteremia other than type III in the presence of a septic picture and an otitic infection, should make one very suspicious of the existence of a pneumonic infection. This has occurred many times in our own experience.

On two occasions we have observed a Streptococcus hemolyticus bacteremia

complicating a pneumococcus type III mastoiditis. The blood cultures were taken because of the development of a septic clinical picture. Both patients had erysipelas. In one instance the erythematous lesion appeared after the blood culture was reported, and in the other case there was a suggestive lesion when the blood was drawn for culture.

The early diagnosis of lateral sinus or primary jugular bulb thrombosis, before the manifest and evident clinical picture has appeared, and the consequent early operation, will contribute greatly to diminish the morbidity, metastases, and mortality of this disease. It is our opinion that adequate and complete blood culture studies with their proper and full interpretation will help to achieve this end to a gratifying degree.

PATHOLOGY

A knowledge of the anatomic relationship of the venous sinuses to the temporal bone and a thorough understanding of the basic pathology of sinus thrombosis is most essential for the proper care of the patient afflicted with this condition. It must be fully realized that the sigmoid sinus and its tributaries are subject to marked variations in size, position and relationship to adjacent structures. There is perhaps no part of the human anatomy that presents such anatomic variations and anomalous conditions as do the venous sinuses. Various types of these have been described by Körner,[6] MacEwen,[7] Eagleton,[8] Ruttin (quoted by Sutherland), and others, and more recently by Ersner and Persky,[9] Furstenberg[10] and Sutherland.[11]

From the practical standpoint it is obvious that involvement of such anomalous veins will have a definite bearing upon the symptomatology and upon the surgical treatment. This is particularly true as regards the blood supply to the head and the question of jugular ligation.

Methods of Extension. Thrombophlebitis of the sigmoid sinus, as is well known, is usually secondary to disease of the mastoid process. Extension to the sigmoid sinus takes place by direct contiguity of the infectious process in the bone or by way of the infected capillaries communicating with the sinus, or by a combination of these pathways. Disease extending to and involving the sinus plate and venous wall (perisinus abscess) is not an infrequent finding. However, the presence of an intact sinus plate does not preclude the possibility of a perisinus abscess or even a thrombophlebitis. Neither the severity of the disease in the mastoid nor the appearance of the sinus plate can always be used as a guide as to the condition of the underlying vein. Thus, in spite of the fact that at operation there may be disclosed little evidence of disease in the mastoid with an intact sinus plate, a thrombosis in the sigmoid sinus may nevertheless exist. This can be explained on the basis that the sinus may be involved secondarily to an extension of thrombophlebitis of its tributaries. It is also true that the appearance of the venous wall is not always an indication as to the pathologic condition within the vein. An apparently normal vein may have within its lumen a well organized thrombus. It is perhaps advisable at this time to emphasize the point that thrombosis of the sigmoid sinus may exist in the absence of physical signs and symptoms. Cases have been reported and we have seen instances in which thrombosis of the sigmoid sinus or jugular bulb was accidently discovered at operation or post-mortem.

The sigmoid sinus at the time of operation is usually described as being dull gray, lusterless, thickened, covered with granulations or necrotic. At times the vessel is found to be collapsed and devoid of blood. These findings naturally would vary, depending, among other factors, upon the severity of the infection in the mastoid, the method of propagation to the sinus, the duration of the illness and the reparative processes. The various theories relative to the formation of a thrombus need not be discussed at this time.

In a general way it can be stated that thrombosis usually takes place at the site of a localized area of phlebitis. The coats of the venous wall are successively infected from without inward. When the intima is involved a parietal thrombus is formed. It is important to emphasize that alterations in the endothelial linings of the intima are prerequisite for thrombosis. The adventitia and media may undergo extensive necrosis with abscess formation as a result of which the lumen of the vessel is considerably narrowed. In spite of this there may be no evidence of thrombus formation. A parietal thrombus naturally increases in size as the infection progresses. When it grows in breadth and occupies the entire lumen it becomes obliterating. A parietal thrombus, on the other hand, may grow lengthwise, organize and heal as such. A thrombus may grow in both directions but its growth is usually more pronounced in the direction of the blood current than in the opposite direction. Thus, histologic examination of fifteen cases of sinus thrombosis which came to postmortem, disclosed the interesting observation that the jugular bulb was the site of thrombophlebitis in every instance. In the vast majority of these cases this condition was secondary to an extension from a diseased sigmoid sinus. While extension of the thrombophlebitis in the opposite direction (retrograde) was not infrequent, in only three cases was the torcular Herophili involved. It is evident that the extent and degree of inflammation may vary from a mild localized area of thrombophlebitis to one of advanced necrosis extending from the jugular bulb or jugular vein to a point beyond the torcular. As was previously pointed out, the extent of thrombosis does not correspond to the area of demonstrable phlebitis. The thrombus may extend beynd the limits of the phlebitis, whereas at times the reverse condition is observed. These facts must be taken into consideration in the evaluation of signs and symptoms and in the surgical treatment. It is perhaps timely to point out that the process of thrombus formation is in reality a protective mechanism on the part of nature to localize and wall off the infection in the blood vessel.

Jugular Bulb Thrombosis. As is well established, extension of infection to the sigmoid sinuses does not always take place via the mastoid process. The sigmoid sinus may be involved by means of retrograde propagation of the jugular bulb thrombosis. The latter condition is usually the result of an extension of the infection in the hypotympanum. Anatomic variations of the bulb, such as an unusually thin, high arched dome or the presence of dehiscences are considered predisposing factors toward its involvement. The infection in the hypotympanum may also reach the bulb by way of communicating capillaries of the submucosa and by direct contiguity with necrosis of bone. Similarly the infection may spread from the Eustachian tube directly to the bulb. When thrombosis of the jugular bulb is secondary to an infection within the tympanum, the resultant condition is known as primary jugular bulb thrombosis, in contradistinction to secondary involvement of this structure from thrombosis of the sigmoid sinus.

It is usually stated that the diagnosis of primary bulb thrombosis should not be made in the presence of a mastoiditis, since under these circumstances, the original site of thrombophlebitis in the vein cannot always be ascertained. There is no question in our minds that primary bulb thrombosis can occur even in the presence of mastoiditis. This has recently been demonstrated histologically by one of us (J. G. D.).[11a] It explains the not infrequent observation at operation of an apparently normal sigmoid sinus in the presence of an otitic bacteremia. When the sigmoid sinus is involved, evidence of generalized blood stream infection usually manifests itself approximately ten to eighteen days after the onset of an otitis. With thrombosis of the jugular bulb, however, the signs and symptoms may appear at a much earlier date, at times within a few days after the onset.

Extension of the thrombophlebitic process from the sigmoid sinus and jugular bulb to the superior and inferior petrosal sinuses is not an unusual occurrence. While this observation is made at operation, the condition is more frequently encountered on histologic examination. It must be borne in mind that the superior or inferior petrosal sinuses may be involved directly from a suppurative process in the petrous pyramid. Owing to the close relationship between the superior and inferior petrosals and the cavernous sinus, it is rather surprising that extension to the latter structure is not more frequently observed. In only one instance was cavernous sinus thrombosis secondary to such extension encountered. A marked engorgement with thrombophlebitic changes in the carotid plexus of veins, however, is a relatively frequent finding. The close proximity of the jugular bulb to the carotid canal with its rich anastomosis is probably another factor contributing to this extension to the carotid plexus.

The emissary vein, because of its close anatomic relationship with the mastoid and with the sigmoid sinus, and because of the frequency of its involvement in thrombosis of the lateral sinus, is worthy of consideration. Thrombosis of this vein may be the result of retrograde extension from the sigmoid sinus or it may be primary, resulting from direct infection in the mastoid. When it is secondary to disease in the mastoid and when it remains localized, it seldom gives signs or symptoms of generalized invasions. However, in the event that this vein is of unusually large caliber, as is occasionally the case, or when extension of the inflammatory process of the vein to the sigmoid sinus takes place, the signs and symptoms of bacteremia will be manifested.

Intracranial Complications. Histologic examination of the temporal bones of patients who have succumbed to sinus thrombosis frequently reveals evidence of petrous pyramid suppuration. This is of considerable importance since the suppurative process in the petrous pyramid may not only be the causative factor in the production of the sinus thrombosis, but may also produce a meningitis by direct extension. A lesion in the sublabyrinthine region of the petrous pyramid in the form of a peribulbar abscess is not an unusual observation. Extension of this lesion to the ninth and tenth nerves, the labyrinth, the carotid canal and the meninges can also be demonstrated.

A suppurative process in the labyrinth is not infrequently encountered. The close relationship between the jugular bulb and the labyrinth predisposes to infection of one from the other. This complication was observed in four of the temporal bones. A causal relationship between the sinus thrombosis and the labyrinthitis could not always be established.

Meningitis is undoubtedly the most frequent and the most dreaded complication of sinus thrombosis. The presence of meningeal signs and symptoms, as is well known, is a frequent clinical observation. These are undoubtedly due to the localized reactive changes of the meninges.

On histologic examination these inflammatory reactive changes of the meninges with cellular infiltration are clearly demonstrable. The dura in the region of the sinus shows varying degrees of thickening with cellular infiltration, localized extradural and subdural abscess and occasionally there is intramedullary suppuration. The exact pathway of extension to the intracranial contents cannot always be ascertained. The most likely routes of extension are: (1) through the blood stream; (2) by way of multiple tributaries which course through the subarachnoid and sudural spaces; and (3) by direct extension through the visceral wall of the sinus or jugular bulb. Not in all cases is the meningitis secondary to the lesion in the sinus per se: it nay be secondary to an associated lesion in the temporal bone. We were particularly impressed with the histologic observation, that there frequently exist pathways of extension leading from the suppurative process in the petrous pyramid directly to

the meninges. The practical importance of thoroughly eradicating such a focus cannot be overstressed.

Repair. Reparative changes in the sinus thrombosis usually take place at a very early stage in the disease. Organization of the thrombus may be observed within a few days after the onset of symptoms. Naturally there are many varying and individual factors which enter into this question of repair. The severity of the infection, the site of the lesion, the removal of the original focus in the bone and the general condition of the patient. Under favorable circumstances the organized thrombus assumes a fibrotic character, becomes less cellular, but more vascular and canalization of the thrombus finally takes place.

SYMPTOMATOLOGY

Many textbooks describe symptoms of lateral sinus thrombosis which are termed classical, but as Tobey,[12] in 1921, and many others have cautioned, these should not be regarded as typical. Tobey repeats, "there is no typical symptomatology." The symptoms resulting from disease affecting the lateral sinus do not differ in essence from those accompanying bacteremias of non-otitic origins. The history of an antecedent middle ear infection regardless of how slight or insignificant it may have appeared to the patient or parent is very important in evaluating the clinical picture of lateral sinus thrombosis. We recall many cases in which careful questioning elicited information from the parent of a preëxisting otitis with pain for only a few hours which was the clue that ultimately led to the proper differential diagnosis. In some of these instances there was no middle ear discharge and when the child was admitted to the hospital the middle ear presented no evidences of recent inflammation.

At times sinus thrombosis in children presents a somewhat different symptomatology from that ordinarily seen in adults and in elderly people with general constitutional diseases, i.e., diabetes, nephritis and arteriosclerosis.

1. *Sensorium.* The patient in the early stages of the disease generally looks alert, bright and may be euphoric. In the first few days they look surprisingly well despite the wide excursions in temperature. Sinus thrombosis may begin with delirium which, after persisting for some time, may eventuate in a psychosis. The delirium has been described as simulating the symptoms of belladonna poisoning. Occasionally the sensorium is clear, the patient looks sick and may hold his hand over the side of his head in the "brain abscess" attitude, and complain of headache.

2. *Chills.* In about half the cases shaking chills occur, but they are not so frequent in children as in adults. The occurrence of more than one shaking chill in twenty-four hours is not uncommon in sinus thrombosis. Politzer[13] states that in the late stages of the disease chills are much more common, so that often several chills are observed daily. Tobey believes that true rigors occur in less than 50 per cent of the cases. In our experience the occurrence of chills has been significant and of considerable importance diagnostically. It cannot be said that the absence of chills militates against the diagnosis of infection in the lateral sinus. In a case suspected of sinus thrombosis more than one chill or more than a single rise of fever within twenty-four hours has always seemed to us to be due to an infectious focus within the circulation.

We know that the type and virulence of the organism, the presence of an obliterating thrombus, diabetes or other debilitating states may be factors in the alteration or suppresion of symptoms despite extensive involvement of the lateral sinus. We feel that when few bacteria are entering the general circulation there may be no body reaction and hence no chills. The resistance on the part of the host to the general invasion of bacteria and their toxins is often responsible for a shaking chill. Absence of chill in sinus thrombosis may

mean that the ends of the thrombus at that time are sterile and no infection is entering the circulation. We recall an instance in which a violent shaking chill lasting many minutes resulted during manipulation of the jugular bulb on the operating table.

3. *Fever.* At various times the literature has shown groups and divisions of cases of sinus thrombosis on the basis of temperature charts.

First, the temperature may be sustained at a high level for a time and only after several days do the characteristic fluctuations occur. Frequently the type of temperature curve rather than the height to which the temperature rises suggests a bacteremia. This is especially true in aged debilitated diabetics and in type III pneumococcus infections. Following a sudden rise in temperature there may be a fall to subnormal. Profuse sweating may occur. However, there are great variations in the temperature curves in different individuals. These curves were termed by Tobey: (a) septic, (b) typhoidal, (c) latent, and (d) no fever.

Only recently we observed a patient with an obdurating thrombus extending from the bulb to the torcular Herophili. Following ligation of the jugular, removal of the obturating thrombus, excision of the parietal wall of the vein, dakinization of the jugular bulb, the temperature subsided. The patient was apparently getting well, walking about the ward. After twenty-three days there was a rise in temperature, with meningeal signs and subsequent exitus. This patient had normal temperature for twenty-three days after her last operation despite the fact that at post-mortem the thrombosis had extended through the torcula to the opposite lateral sinus. One may frequently be misled in elderly people, diabetics, in type III pneumococcus infections, in severe nephritis, if too much significance diagnostically is placed upon the extent of temperature fluctuations.

4. *Pulse.* At the onset the pulse is rapid, bounding, and not easily compressible, and it corresponds to the temperature. As the temperature becomes higher there usually appears a proportionate increase in the pulse rate. With progression of the sepsis a disproportion between the pulse rate and temperature becomes evident, indicating a poor prognosis. In an undamaged myocardium with elevation in temperature there results an increase in the pulse rate. A persistently slow pulse or a pulse which becomes slow after a rigor with attendant rise of fever, is of good prognostic import.

5. *Fundus Changes.* As described in an earlier paper, fundus changes, i.e., hyperemia, neuritis and papilledema were observed many years ago. Of late the changes in the fundi in sinus thrombosis have received increased attention and from the available statistics in the literature it appears that these changes occur in anywhere from 10 to 16 per cent of the cases. Retinal congestion is not considered a change in the fundus as is blurring of the disc margins and measurable papilledema. Observers have noted that increases in the number of diopters of papilledema may result many days after operation. Progressive choking of the disc after operation is not necessarily a sign of grave import. We have frequently noted the development and increase in papilledema lasting weeks and months after adequate operation with complete recovery and normal vision. Frequently when papilledema is noted before operation, it increases even after adequate surgical measures have been taken which control the bacteremia. No satisfactory explanation for this has been offered. Papilledema after sinus thrombosis may be present and even become more marked without any other signs or symptoms of increased intracranial pressure. Sometimes, however, the manometer will show increase in the cerebrospinal fluid pressure.

The eye grounds are examined routinely and occasionally they present the first change which will direct suspicion to the lateral sinus. The presence of fundus

changes is suggestive, but the absence of fundus changes does not preclude the possibility of involvement of the lateral sinus. Nor is there any definite relationship between the extent of the disease (obturating sinus or phlebitis) and the fundus changes.

6. *Local Signs.* In our experience we have been impressed by the frequent presence of a very hard tender gland at the angle of the jaw on the involved side. Tenderness along the course of the internal jugular vein, in the absence of inflamed cervical glands, may be of some significance. Occasionally one can feel a cord-like vein which is tender and can be rolled under the finger in thin patients. We do not recall that enlargement of the external jugular was ever observed or was of any clinical importance. On the other hand dilatation of the veins of the scalp has been observed several times.

The sign described by Griesinger,[14] namely edema and tenderness over that area drained by the mastoid emissary vein, is occasionally found, but is probably more significant of perisinus abscess than sinus thrombosis. Many instances of complete obturation of the lateral sinus and emissary vein were encountered in our series of cases but rarely the Griesinger sign. According to Voss[15] a venous bruit is heard over a normal internal jugular vein which is not audible in sinus thrombosis with an occluded internal jugular vein.

The Tobey-Ayer[16] test is often of value in cases of bilateral mastoiditis in which a bacteremia exists and where the lateral sinuses after exposure offer no clue as to the affected side. Alternate pressure on the veins, however, should be repeated several times before drawing any conclusions. Furthermore, due consideration to anatomic differences should be given. Hilding[17] described a false response to jugular compression due to an anomaly of the lateral sinus. Woodhall[18] discussed the variations of the cranial venous sinuses and the validity of the Queckenstedt test in the diagnosis of lateral sinus thrombosis. When an otitic sepsis is suspected, the complaint of pain on swallowing without any apparent local cause may mean disease of the jugular bulb, with inflammatory perineuritis of the glossopharyngeal nerve in its passage through the jugular foramen.

Widespread thrombosis with extension into the facial vein results in edema of the face and eyelids. When extension into the superior longitudinal sinus occurs, the veins of the forehead and edema of the scalp become prominent. Extension of the thrombus into the cavernous sinus of the same and opposite sides, and into the lateral sinus of the opposite side occasionally takes place.

7. *Other Associated Findings.* With surprising uniformity we have found a palpable spleen as the sepsis progresses. The spleen may enlarge to three fingerbreadths below the costal margin. With subsidence of the sepsis the spleen gradually becomes smaller. We have seen unilateral and bilateral external rectus palsy.

Daily hemoglobin determinations reveal a progressive anemia and the white blood counts and differentials most frequently show a leucocytosis of varying degree, with significant changes in the cytoplasm of the leucocyte. Rosenwasser and Rosenthal[19] and others have shown that study of the blood with especial regard to the toxic granules will yield more accurate information than will merely a consideration of the leucocyte count and a modified Schilling hemogram. Occasionally there is a leucopenia with otitic invasions.

Metastasis. The large joints, the hip, ankle, elbow and shoulder are the most common sites for metastatic foci. In children the hip joints seem to be the site of election. Generally the lesion is in the periarticular tissues rather than in the joint proper. Abscesses in the soft parts about joints in the buttocks and thigh also occur. The lungs are frequently involved. We recently observed a case of sinus thrombosis reported by one of us (H. R.[20]) in which a pleuropulmonary suppuration was evident at the end of several days.

Early pulmonary lesions have been described by Lillie[21] and others. It is surprising that pulmonary suppuration is not more common in sinus thrombosis when one considers the large numbers of bacteria that must be filtered out in the lung bed.

Dissolution of bone with extension of infection ex contiguo to the sinus takes time and therefore the time relation between the onset of the otitis media and the recognition of metastatic foci is significant. On the other hand, there are cases (primary bulb thrombosis) in which the extension of infection does not postulate bone destruction but rather extension by way of preformed pathways (blood vessels or dehiscences) or a thin hypotympanum. In these instances metastases may become evident shortly after the beginning of the middle ear inflammation.

We do not believe that any definite conclusions can be drawn as to the significance of the apparently increased number of metastases following jugular ligations reported by some authors. Metastases are widespread in an otitic bacteremia, and foci which may have been deposited early in the bacteremia need not become clinically evident until some time later. In other words when a metastatic focus becomes clinically diagnosed is no reliable indication as to when it was first laid down. In our experience we have found no increase in the number of metastatic foci in these cases in which jugular ligation was employed, as compared to the cases in which it was not employed. A latent metastasis may become evident days and even weeks after its original deposition. Fenton[22] and others have discussed delayed metastases and have similar viewpoints.

Some of the less common metastatic lesions are ophthalmia (Sonnes[23]), thyroiditis (Collet[24]), endocarditis (Libman[25]) and carbuncle of kidney. Metastases alter the ultimate prognosis and prolong the morbidity. Neglected cases are more apt to develop metastases, probably because the bacteremia has been allowed to continue longer. We have not seen such metastases as occur from infectious lesions on the arterial side of the circulation and we use this clinical observation in making a differential diagnosis when necessary. In sinus thrombosis we have not observed white-centered petechial conjunctival or skin lesions so common in acute or subacute endocarditis.

DIAGNOSIS

The typical classical picture of sinus thrombosis presents no difficult problem in diagnosis. On the other hand, coincidentally with or shortly after middle ear suppuration or mastoiditis, there occasionally occur clinical pictures which may simulate sinus thrombosis but which subsequently prove to be either acute endocarditis, pneumonia or erysipelas.

In uncomplicated lateral sinus thrombosis we have never seen petechiae, whereas this particular embolic lesion is frequently noted in acute endocarditis. The changing character of the cardiac murmur from day to day, embolic lesions in the ocular fundi, the history of previous rheumatic fever and finally the presence of large and almost equal numbers of organisms in the blood cultures taken simultaneously from the internal jugular vein and the peripheral brachial veins (Ottenberg) all, according to our experience, definitely point to acute endocarditis rather than sinus thrombosis.

One of us (I. F.) was called to see a patient in consultation some years ago in which the following had already transpired: The patient, a young man, had had a mastoiditis and following the operation had a chill with a rise in temperature to 104°F.; the lateral sinus was obliterated and the internal jugular vein ligated, but the septic course continued. There was no evidence of involvement of the lateral sinus. Culture of the blood was found to produce type 1 pneumococcus. At this stage of the illness, the patient was seen in consultation and early signs of pneumonia could be detected. That the chest must be carefully examined clinically and roentgenologically if necessary, needs no

repetition. The fact that pneumococci other than the type III do not cause lateral sinus thrombosis was the important diagnostic point which, if realized, might have saved this already sick young man an extensive, unnecessary and dangerous operation.

Occasionally two to five days after mastoidectomy a patient may have a shaking chill with a rise in temperature to 104 degrees or higher. There may be present a fine exhaustible nystagmus and the patient may appear very sick. The wound should promptly be inspected to see whether there is any local evidence of an early erysipelas. In a certain percentage of cases of erysipelas we have noted nystagmus twelve to twenty-four hours before the skin lesion appeared. Blood cultures at this time are usually negative although occasionally there may be a transitory bacteremia. Erysipelas does not cause a persistent bacteremia. Spread of the erysipelas frequently is associated with sharp spike-like elevations in temperature and may simulate the temperature curve of otitic sepsis.

Not infrequently, after the primary focus in the lateral sinus is adequately dealt with, the septic temperature may be maintained by a metastatic deposit in the pleuro pulmonary tissues in or about a joint or in the meninges but these lesions do not present diagnostic problems for any length of time. Metastases are not responsible for a persistent bacteremia.

TREATMENT

Many of the older concepts governing the treatment of lateral sinus thrombosis, have been the subject of more or less continued discussion and criticism in recent years and as a result of this continued interest many phases of the treatment of this condition have been clarified. The usual accepted treatment of lateral sinus thrombosis embodied exposure of the lateral sinus sufficiently to encompass the limits of disease, ligation or at times excision of the internal jugular vein, opening the lateral sinus with removal of the thrombus when present, excision of the parietal wall and the establishment of free bleeding from the bulb and the torcular end. This time-honored surgical approach directed to exclude the suppurative focus from the general circulation, still forms the basis of our present treatment.

The care of a patient ill with sinus thrombosis presents many problems, one of which is when to operate. On many occasions patients have been admitted to the otologic service of the Mount Sinai Hospital obviously suffering from a severe otitic sepsis. In our experience we have found it better to postpone surgical intervention for a few hours or even a day and to institute general supportive measures, such as transfusions and continuous intravenous, until improvement in the general condition of the patient results. At times we defer operation in the presence of hyperpyrexia. When general improvement is noted, and during the interval when the temperature is lower, operation is performed. Operation and its attendant shock frequently may be the contributing cause of death in these extremely sick patients. When operation is resorted to we do not hesitate to stop at any time during the operation should the general condition of the patient warrant it. On a number of occasions we have had to do this and have had no reason to regret this course. It is a scant comfort to the surgeon to know that a complete operation has been performed but to lose the patient because there was superimposed upon the sepsis a prolonged operation and anesthesia with their resultant shock.

Another problem which has aroused much discussion is what to do with the internal jugular vein in lateral sinus disease. From the very beginning of lateral sinus surgery the necessity for ligation of the internal jugular vein has had its protagonists and antagonists. The reasons advanced are too well known and need not be repeated. A few brief examples in specific instances will explain our view.

Generally speaking in those instances in which there is grave doubt as to the advisability of leaving the internal jugular vein patent, we ligate it. However, even in some instances in which the bulb was thrombosed, we have temporized and not immediately ligated the jugular vein. In those cases in which the bacteremia continued, we have subsequently ligated the vein. In a considerable number, however, operation on the lateral and sigmoid alone was sufficient to control the general invasion. In those cases in which there is a mural thrombus or phlebitis with free bleeding from the bulb, we believe it is safer to ligate the internal jugular vein. We have not found it necessary to excise the internal jugular vein. In only two cases in many years was excision resorted to and this in patients who were admitted with evidence of extensive involvement of the internal jugular vein. At operation the thrombosis extended into the innominate vein in both cases. One patient recovered after excision of the internal jugular vein with ligation close to the subclavian.

We pass a double ligature around the vein but do not cut between the ligatures as some advocate, because we believe fewer neck infections result in this way. The ligatures are separated one from the other by about 1 inch. In those occasional cases in which suppuration in the neck occurs, it has not been serious. It has not been necessary to use the procedure of Alexander, of double ligation of the vein with suture of the upper end of the vein to the skin and irrigating the bulb from above. If the thrombus in the bulb breaks down, the bulb is irrigated daily or even twice daily with Dakin's Solution. It is not uncommon to note prompt fall in the fever when locculated collections of pus in the bulb are treated in this manner.

Just before operation on the lateral sinus the patient routinely receives a continuous intravenous, the rate of which can be increased or slowed depending on the general condition, rate and volume of the pulse, etc. We are opposed to hurried intravenous therapy after it is noted that the general circulation is failing, when the use of this therapy preliminary to and during operation frequently forestalls this complication. Preliminary to operation the patient is always grouped, and transfusion may be given by way of the continuous intravenous. We are favorably impressed with the work of Baum,[26] Gill,[27] and others in using immune donors whenever possible. Unfortunately we have had little experience along these lines.

There is today an additional aid in the treatment of sinus thrombosis, namely sulfanilamide. Its use is avoided until a diagnosis is established. Then it is used as an adjuvant to surgical therapy. When the drug cannot be administered by mouth, prontosil 8 per cent is given intravenously. When using sulfanilamide we attempt to raise the blood concentration of this drug rapidly to as high as 15 mg. per 100 c.c., especially if there are meningeal signs. When this drug is used prematurely we find it is apt to mask the clinical picture somewhat and therefore the latter must be evaluated bearing this in mind.

REFERENCES

1. Libman, E. *Tr. Am. Otol. Soc.*, 10: 315, 1906.
2. Friesner, I. Infections of the middle ear. *Arch. Otolaryng.*, 4: 256–267 (Sept.) 1931.
3. Schwartzman, G., and Goldman, J. L. Streptococcus hemolyticus bacteremia. *Arch. Surg.*, 34: 82 (Jan.) 1937.
4. Goldman, J. L., and Shwartzman, G. Streptococcus hemolyticus bacteremia. *Ann. Otol., Rhin. & Laryng.*, Dec. 1935.
5. Maybaum, J. L., and Goldman, J. L. Primary jugular thrombosis. *Arch. Otolaryng.*, 17: 70 (Jan.) 1933.
6. Korner, O. Die otitische Erkrankungen des Gehirns. Weisbaden, 1902. J. F. Bergmann.
7. MacEwen, J. quoted by Eagelton.[8]
8. Eagelton, W. P. Circulatory disturbances following ligation of the internal jugular vein. *Arch. Otol.*, 35: 91, 1906.
9. Ersner, M. S., and Myers, D. Intracranial complications from venous circulatory disturbances in the lateral sinus and jugular foramen. *Laryngoscope*, 43: 800 (Oct.) 1933.
10. Furstenberg, A. C. Septic thrombophlebitis of the sigmoid sinus. *Tr. Am. Ac. Opbth. & Otolaryng.*, panel discussion, 1937.
11. Sutherland, J. M. Otitic sinus thrombosis. *Arch. Otolaryng.*, 27: 1–34 (Jan.) 1938.

11a. Druss, J. G. Thrombophlebitis of the sigmoid sinus. *Arch. Otol.*, in press.
12. Tobey, G. L. Lateral sinus infections. *Tr. Am. Laryng., Rhin. & Otol. Soc.*, 1921, pp. 201–213.
13. Politzer's Diseases of the Ear. Phila., 1926. Lea & Febiger.
14. Griesenger, W. Fortgesetzte Beobachtungen über Hirnkrankheiten. *Arch. d. Heilk.*, 3: 446, 1862.
15. Voss, O. Ein Beitrage zur Operativen Behandlung der Sinus Thrombose. *Arch. f. Obrenheilk.*, 39: 89–101, 1895.
16. Tobey, G. L., and Ayer, J. B. Dynamic studies on the cerebrospinal fluid in the differential diagnosis of lateral sinus thrombosis. *Tr. Am. Otol. Soc.*, 17: 174–188, 1925.
17. Hilding, A. False response to jugular compression due to anomaly of lateral sinus. *Arch. Otolaryng.*, 26: 143–145 (Aug.) 1937.
18. Woodhall, B. Variations of cranial venous sinuses. *Arch. Surg.*, 33: 297–314, 1936.
19. Rosenwasser, H., and Rosenthal, N. The blood picture in otitic infections. *Arch. Otolaryng.*, 14: 291–308 (Sept.) 1931.
20. Rosenwasser, H. Unusually early pleuropulmonary suppuration in sinus thrombosis. *Laryngoscope*, 46: 519–522 (July) 1936.
21. Lillie, H. I. Thrombosis of superior longitudinal or straight sinus by extension from the lateral sinus. *Ann. Otol., Rhin. & Laryng.*, 36: 324–331 (June) 1927.
22. Fenton, R. A. Lateral sinus thrombosis with delayed metastasis. *Ann. Otol., Rhin. & Laryng.*, 36: 675–677 (Sept.) 1927.
23. Sommer, I. Ueber metastatische ophthalmie bei otogener Sinus Thrombose. *Monatschr. f. Obrenheilk.*, 63: 419–427 (April) 1929.
24. Collet, F. J. Thyroïdite suppurée compliquant une phlébite du sinus latéral. *Lyon méd.*, 143: 673–676 (June) 1929.
25. Libman, E. A further communication on the importance of blood cultures in the study of infections of otitic origin. *Internat. Otol. Congr. Tr.*, 9: 127–176, 1912.
26. Baum, H. L. A method of specific treatment in certain streptococcic infections. *Arch. Otol.*, 20: 504–512, 1934.
27. Gill, E. G. Management of blood stream infection with special reference to immunized blood transfusions. *South. M. J.*, 30: 496–502 (May) 1937.

Little is known of postmortem examinations before the end of the thirteenth century, when, during a plague in Italy in 1286, a physician of Cremona opened many bodies to discover the cause of the pestilence.
From—"The Postmortem Examination" by Sidney Farber (Thomas).

THE DIAGNOSIS AND TREATMENT OF OTOGENIC MENINGITIS

SAMUEL J. KOPETZKY, M.D., F.A.C.S.

Professor of Otology, New York Polyclinic Medical School

NEW YORK CITY

INTRODUCTORY

THE developments in successfully mastering the problems presented by otogenic meningitis have developed very gradually. So slowly has the evolution in management and therapy been brought about, that it is impossible to date the time when the pessimistic prognosis in such cases gave way before the advent of the prevalent optimism. Not so long ago a diagnosis of a bacterial otogenic meningitis was invariably followed by a fatal outcome.

Efforts were strongly directed toward evolving signs and symptoms for early diagnosis. As earlier and still earlier signs and symptoms came to be recognized, a differentiation between an invasion of the meninges and an infection of these tissues became possible. The recognition of that phase which I term that of invasion, logically led to a study of the source whence the invasion came.

With the recognition of the focus of origin, if it could be localized, a better comprehension of the focal bone lesions in the temporal bone causing the invasion of the meninges resulted. A whole series of intercurrent, or penultimate bone lesions came to be recognized, and surgical therapy applied to their eradication. There then were obtained observations which demonstrated that even in the face of a meningeal invasion by pathogenic bacteria from such a penultimate bone lesion, when the clinical situation thus presented is comprehended and the sickened bone is promptly removed, the meningeal invasion abated; and a return to normalcy in the meninges could invariably be expected.

It was further established that the tissue reactions in the meninges, with only slight modifications, were almost always similar, irrespective of the nature and the kind of bacterial agent which was the pathogenic factor in the given case. Except for specific antitoxins and bactericidal sera, and the clinical recognition of specific manifestations from some given organism, the recognition of the actual bacterial causative agent became less important than the recognition that in otogenic meningeal cases there also exists a bone lesion from which the cranial cavity contents became invaded and infected.

The diagnosis of otogenic meningitis for a very long period was made upon an anatomic basis. Its clinical aspects colored its designation. Spinal puncture and examination of the cerebrospinal fluid had long been known and practiced, and the clear fluid obtained in the early stages, followed as it inevitably was—as the lesion in the meninges progressed—by purulent fluid, served to give diagnostic terminology. The terms, serous meningitis, purulent leptomeningitis, circumscribed meningitis, have in modern otology less value because they give no point or clue to successful therapy. While the use of these terms was in otologic vogue, little progress in securing recovery was made.

Likewise, because the knowledge of the physiology of the cerebrospinal fluid was meager and a bacteriogenic infection was mooted, the establishment of drainage of the meninges was attempted. Because surgically, drainage is successful in most abscess formations, it was believed that abscess formation, modified by the tissues of the meninges, was the significant lesion. Surgical drainage of the meninges proved disappointing, and is now generally abandoned, (1) because abscess formation

actually does not take place; (2) because we are mostly dealing with a circulating fluid. The opening of the cerebrospinal spaces to establish drainage, if the drainage channels remain open beyond twenty-four hours, so disturbs the cerebrospinal fluid circulation, that the specific physiologic function of this body fluid is seriously modified. Then the patient not only suffers from his meningeal infection and its concomitant tissue reactions, but in addition he suffers from the disturbed physiologic action resultant from this, with cerebrospinal tissue metabolism disturbances added to the clinical picture.

The modern otologist therefore is content to view the meninges as a whole; to seek diagnostic data of each phase of their involvement in pathogenic lesions, to comprehend the rôle which the inciting focal bone lesion plays in their development, and, unless surgically removed, in their persistence. He must have a working knowledge of the chemistry of the cerebrospinal fluid in health and in disease, particularly pyogenic infections of the meninges. Lastly he must comprehend the rôle played by the products of brain cell activity as they are emptied into the circulating cerebrospinal fluid, and the deleterious effects which failure in the removal of these brain cell products cause to the brain and to the system of the patient generally.

ELEMENTS IN DIAGNOSIS

In estimating factors which the given patient presents to make a diagnosis, certain physiologic functions are taken into account. Basically the factors in diagnosis come from the clinical picture and from a study of the contents of the cerebrospinal fluid obtained through spinal puncture.

In the earlier day, before the knowledge of these finer differentiations had been won, the clinical diagnosis of meningitis rested on the evidence of a disturbed sensorium, a high fever, nuchal rigidity and marked opisthotonos, the inhibition of various nerve functions; varying degrees of gastric disturbances, such as vomiting and refusal to take food; and central nervous system reactions, such as interference with the vasomotor and respiratory centers. Headache and photophobia during the stage of consciousness were constantly present symptoms. Changes in the optic discs gave important signs also. When these symptoms were present a confirmatory spinal tap, revealing a cloudy fluid with pathogenic bacterial content, substantiated the diagnosis.

Efforts were made at differential diagnosis from a study of the cytology of the fluid, from its albumin and globulin content, and from the flocculi which formed in the test tube containing the fluid after a period of standing. Of course, search for bacteria and their growth on artificial culture media were also in vogue.

At this period in our knowledge, the clinician was never in doubt as to the diagnosis of meningitis. The clinical evidence of it was explicitly apparent. Cases were seen, in which the prodromal phase of the lesion passed asymptomatically. Patients were sometimes ambulatory, and suddenly the terminal phase of their lesion presented itself clinically, and these were termed fulminating cases of meningitis. In the short terminal phase, the whole bedside findings left no doubt of the diagnosis, and in many cases the subsequent autopsy confirmed it.

In the search for earlier and still more early signs of meningeal infection, the prodromal phase of the lesion gradually came under study, and it is exactly from the findings which the clinical laboratory gives of the spinal fluid contents in this early, almost asymptomatic period that our greatest progress has been won, not only for diagnosis but also for therapy. Of the clinical evidence, only headache and slight sensations of pain on flexing the head onto the chest, and occasionally a very mild photophobia are symptomatic of these early stages in the development of an otogenic meningeal invasion or infection.

The otologist who is on his guard during this prodromal phase, and who utilizes the resources which modern clinical laboratory methods give him, meets less and less with the so-called fulminating type of meningeal infection. Long before that stage of development is reached, he has made his diagnosis—even during the asymptomatic stage—and begun his therapy. As a result, he accomplishes cures which heretofore were impossible. Hence a rather detailed exposition of the laboratory findings in various stages of meningeal involvement becomes important.

The study of the cerebrospinal fluid consists in determining its bacterial flora, knowing its cytology, and making a chemical examination of its contents.

We may be permitted to dismiss the bacterial findings with a very brief note. The search for bacteria should be made as soon as the fluid is withdrawn. In other words, a smear should be made from the centrifuged fluid; a sufficient quantity should be centrifuged to provide enough of a sediment to make the smear. Then, in addition, cultures should be made on various culture media.

There is very little significance in the given case in finding the specific invading microorganisms, because the tissue reactions in the meninges are the same with most of them. Of course, where specific antitoxins or antibacterial sera are available for therapeutic use, the early positive identification of the given organism is of great help in bringing the administration of the specific antitoxin or the specific bactericidal sera into early use. But excepting perhaps the serum to combat the intracellular meningococcus of Wexebbaum, the causative agent of epidemic cerebrospinal meningitis, and a few of the known bactericidal seras for use in some of the types of pneumococcus meningitis, little of great therapeutic value is available to the clinician from the bacterial examination of the fluid. Finally, too often the examination of the fluid for bacteria gives a positive finding only at the time when the clinical picture is clearly manifest. It usually comes too late to be of therapeutic significance. In spite of these factors, the search for and the identification of the bacterial causative agent should never be neglected, but should always be recorded. Taken together with factors later to be discussed, the record of such a search and a negative finding also have significance in estimating the features of a case.

It is in the chemical study of the fluid that the most important diagnostic factors are obtainable. Unfortunately the chemical study of the spinal fluid, while gaining ground and becoming established as routine procedure, is still too often neglected in its entirety and is not generally available everywhere to otologic surgeons. Because of this, I shall stress its findings and their interpretation more strongly, as I believe many are neglecting this means which might help to save lives.

Chemical Studies of the Pathologic Findings in the Spinal Fluid. Naturally, the cytology has already established the presence of cells, indicating a pathologic condition, and their number and kind. In mass they constitute the globulin and most of the albumin of the fluid. The presence of marked amounts of albumin and globulin in the fluid with few cellular elements is rare.

There is in normal spinal fluid a copper reducing substance. In 1912, in a search for an early diagnostic sign to indicate the presence of a bacterial meningitis before the actual presence of bacteria could be factually established, this copper reducing agent was found. The estimation of the loss of the copper reducing carbohydrate in the spinal fluid, until it gradually disappears, is now generally used in establishing diagnostic data. The exact chemical estimation of the carbohydrate present in successive series of spinal taps gives indication of the development of a bacterial infection.

In securing specimens for chemical examination, it has become necessary to study not only the chemistry of the spinal fluid, but also at the same time to study

the same elements in the patient's blood, specimens of which are taken just after the spinal tap is finished. Both sets of specimens are covered with a layer of melted paraffin (of low melting point) to prevent evaporation and other changes. Furthermore, the specimens should be taken in two separate sets: the fluid taken at the tap divided into a portion kept scrupulously aseptic for bacterial analysis, and a second portion to be subjected to chemical examination. The specimens from the circulating blood stream are likewise separated for similar purposes. The finding of a positive bacteremia is of diagnostic significance.

To the carbohydrate content estimation, estimations of the cerebrospinal chlorides, the carbon dioxide content, the lactic acid content, the study of the *p*H of the fluid is added. These elements are also studied in the blood specimens taken at the same time, and the results of the finding in the fluid are compared with the finding in the blood.

In summary, lactic acid in meningitis is found in much increased proportions in the fluid as compared with the amount in the blood. Any patient with a continuing high fever will present an increase in lactic acid in the blood. However, in meningitis the increase in lactic acid in the fluid is much more marked than the increase in the patient's blood. This increase is disproportionate, the ratio of the rise often being three or more to one.

In comprehending the chemical changes taking place in meningitis, one must have a clear understanding of the accepted principles of ionic exchange through physiologic membranes and the devices which are known to aid in maintaining the neutrality of the body fluids. Deviations from the normal may then be evaluated, and the recorded observations of empiric results of many investigators become synthesized and rational.

We know that in a given individual there are more chlorides in spinal fluid than there are in blood plasma. In meningitis this excess of spinal fluid chlorides tends to be eliminated, and the amounts tend to be more nearly alike. There is evident a marked decrease in the bicarbonate content of the spinal fluid; values are often obtained as low as 33 per cent below that of blood plasma.

The *p*H of the spinal fluid from meningitic cases is much lower than either that of normal spinal fluid or that of normal blood. It is also almost always lower than that of the blood plasma of the same individual.

The normal spinal fluid is practically protein free, the protein nitrogen ranging from 35 to 100 mg. per 100 c.c., while the protein content of the plasma ranges from 6,500 to 8,500 mg. per 100 c.c. The Donan laws govern the permeability of all the ions concerned. Of these, all react to the Donan law and are permeable except that of protein.

Clinicians will be less concerned with the details of these factors of the Donan law than with its practical application in the given case. Therefore, it suffices for all our purposes to state and to remember only this: that the ratio of lactic acid in the fluid to that of the blood is normally about 1 to 1, and in meningitis the spinal fluid lactic acid rises so that the relation becomes 3 or more to 1. There is a drop in the chlorides, a drop in the carbon dioxide, and a drop in the *p*H of the fluid from meningitic cases.

Besides these chemical examinations, the physical characteristics of the fluid are studied. The pressure under which it is obtained should be recorded at the commencement and at the end of the tap. There is significance in knowing how promptly the intraspinal pressure drops.

Before proceeding with elements of diagnosis of otogenic meningitis, it becomes necessary to summarize the average findings in otogenic infections wherein the meninges are not the actual seat of the pathology but show reactions because of their proximity to the bone focus. They give evidence of irritative reactions, indica-

tions of which are found in the spinal fluid.

Summarized, they are as follows: In cases of uncomplicated mastoiditis a slight difference is found when the mastoiditis is not accompanied by an upper respiratory infection. The upper respiratory infections and other general infectious diseases bring with them some changes in the spinal fluid. In uncomplicated otitis and acute mastoiditis, the cerebrospinal fluid shows an abnormal cell count, numbering occasionally up to 30; but the fluid is normal chemically, and abacterial, with moderate pressure or normal pressure. The fluid in acute mastoiditis has similar characteristics to that of an otitis, except that a weak protein reaction may be found. With epidural suppurations that have not been present for a long time, or are not of large extent, there is an increase in the number of cells; but the chemistry is normal and there is no bacterial content. In uncomplicated petrosal apicitis, as a rule, the only change noted is an increased cell count, moderately higher than in uncomplicated mastoiditis. The fluid is otherwise normal.

With a large epidural abscess, it is expected and generally found that the fluid is chemically normal and is abacterial; but the intracranial fluid pressure is increased. The protein reaction in about three-fourths of the cases remains normal, while in the others it is mildly abnormal. Cytologically, unless the meninges distinctly react to the presence of the epidural pus collection, the cell count of the fluid is normal. This is true even for a epidural abscess located over the petrosal pyramid tip. If meningeal tissue reactions are set up by the epidural abscess, the findings begin to take on the aspect of a meningitis. There is an increase in pressure, the cell count varies—ranging between 9 and 200, but the protein reaction remains weak. Chemically the fluid remains normal. In cases of otitic sepsis, where there is no demonstrable thrombosis in the large venous channels, the spinal fluid is clear, under moderate pressure, with an increased cell count, but bacteria-free, and chemically almost normal. There are no marked changes. The findings will be almost similar to those found with serous meningitis.

In cases of otitic sepsis, with demonstrable sinus thrombosis, the spinal fluid findings vary individually with each case. In about 50 per cent of the cases, there is increased intracranial pressure due to circulatory changes, and the cytology gives an increase in lymphocytes. The cerebrospinal fluid findings are indicative of a protective meningeal tissue reaction. Where thrombophlebitis occurs in the first few days of an otitic infection there are more marked changes noted in the spinal fluid than if it develops later in the course of the infection. In the early cases, the cell count in the fluid is found increased, and the higher the number of cells found, the more predominantly will the polymorphonuclear forms occur. Chemical abnormalities are also apt to be found. The fluid often will contain bacteria (about 17–20 per cent), and this will usually be the observation in cases that tend to terminate fatally. In brain abscess, the analysis of the spinal fluid will give a picture similar to that obtained from a meningitic case: a high cell count, an abnormal chemistry, and the finding of many bacteria. If the abscess is well walled off, and its presence is denoted only by its pressure signs, either direct or indirect, the fluid may be crystal clear, with only a very slight deviation from the normal chemically, and it may be abacterial, but the pressure is much increased. When a brain abscess leaks or rupture into the meningeal tissues has occurred, the fluid will be found to contain many cells, it will be turbid, and it may contain bacteria. Chemically, too, it will give abnormal findings.

Having summarized the findings obtained from most cases where the focal lesion is located in some part of the temporal bone—in the mastoid process, the tympanic cavity, the venous blood channels, or in the petrosal pyramid—it becomes necessary briefly to summarize the

findings which go to make up the picture which I term a meningeal invasion, and differentiate this picture from the findings of a meningeal infection with tissue reactions.

In meningeal invasions, bacteria reach the cerebrospinal fluid channels, are found in the spinal fluid tap, but meningeal tissue reactions have not yet become established. During this phase of the evolution of the lesion, the demonstration of the presence of pathogenic bacteria in the fluid is of diagnostic and also of therapeutic significance. The fluid is otherwise, however, normal, or deviates only slightly from the normal. There may be a decreased amount of carbohydrate present and some globulin and albumin, but these two latter elements are generally found only in traces. The chloride and the carbonate content, as well as the *p*H, are apt to approximate the normal. The lactic acid ratio and the pressure are slightly increased. Yet the clinical picture may be stormy, and the temperature range high. Clinically the diagnosis of meningitis is substantiated by the neurologic findings, but the analysis of the spinal fluid, as indicated above, denotes an absence of tissue reactions in the meninges, and I term the case, despite its clinical manifestations, a meningeal invasion.

As the case develops—assuming that no successful therapeutic intervention has taken place—the series of succeeding spinal taps reveals a gradual diminution in the carbohydrate content, until a time is reached when it is no longer present. Meanwhile, succeeding series of examinations disclose the positive presence of pathogenic bacteria. Likewise, the succeeding comparisons of analysis of the spinal fluid and of the blood, show the gradual drop of the fluid *p*H, the chlorides and the carbonates. The lactic acid ratio of this element in the blood to that in the spinal fluid, begins to approximate the ratio of 1 to 3 or more. The protein reaction becomes stronger, albumin and globulin are more markedly present, the cell content increases, with polynuclears predominating. The pressure also rises. The fluid is turbid, varying to frank purulency. The clinical picture, which often started with only a persistent, intense headache with some photophobia, develops to show the wonted signs significant of a meningitis. The meningeal invasion has been succeeded by meningeal tissue reactions, and an otogenic meningitis is now present.

The period of invasion may last for days. Clinically the temperature is not very high. The headache is the most consistent sign. The Kernig, too, is often early evident. Mild photophobia frequently occurs during this period. Often the patient lies relaxed, and no bedside evidence is presented denoting specifically an intracranial lesion. If, suspecting the advent of a meningeal infection, one performs a spinal tap, the cell count finding will be startling and cause a search for the focal bone lesion whence comes the meningeal invasion.

TREATMENT

It must be obvious, after a study of the case from its beginning and recognition of the phase of invasion, that the first therapeutic endeavor must be directed toward the eradication of the sources of the invading stream of bacteria. This consists in a systematic search of each and every part of the temporal bone to locate the penultimate lesion (that is, the lesion subsequent to the otitic and mastoidal infection, which developed either simultaneously with these, but remained after surgery had been applied to the tympanic cavity and the mastoid process, or which developed after this primary surgery on the mastoid process). Symptoms are few and very slight, often overshadowed by the symptoms from the acute mastoiditis.

The penultimate lesion is often seated in some part of the petrosal pyramid. It may be found in the channels of the labyrinth, or it may be located in some portion of the osseous perilabyrinthine structures, particularly at the petrosal tip. The symptoms of suppurative petrositis are now comprehended, and the rôle this lesion plays is understood. The involve-

ment of the labyrinth also gives significant symptoms which make their diagnosis easy. Of all these pathologic changes, the ultimate lesion is a meningeal invasion, terminated by a meningeal infection.

Therefore, the first step in therapeutics is the removal of the causal bone focus. This may entail a labyrinthectomy or a thorough surgical exploration of the petrosal pyramid. It is beyond the scope of this paper to detail the various surgical techniques to accomplish these operations. Here it suffices to note them, and recall that these means of surgical intervention, promptly instituted when the lesions are recognized, have often been adequate to end the stage of invasion and prevent the formation of the ultimate lesion which would have developed. In other words, the otogenic meningitis is, in a sense, aborted.

Often the otologic surgeon has no opportunity to study the case from its commencement. Often it comes to him late, when symptoms of meningitis are already strikingly present. Often one sees such cases after simple mastoidectomy has been performed by others. The first problem then is again the immediate determination, as far as that is possible, of the stage the intracranial lesion is then presenting. An estimation of whether one is faced with a fully developed meningitis with tissue reactions existing must be made. It must also be determined whether the patient has most or all the clinical signs of a meningitis, but actually has an undiscovered penultimate lesion accompanied by a meningeal invasion. In either case therapy must be prompt. Decisions must be promptly made if the patient is to be saved.

It is my custom, when faced with any case having clinical symptoms of otogenic meningitis, immediately to transfuse the patient. A few notes regarding whole blood transfusions are warranted because of the important rôle this therapeutic measure holds in the treatment of meningitis.

Whole blood transfusion has a distinct influence on the chemistry of the cerebrospinal fluid. During the stage of invasion, when the patient may even present bacteria in the fluid, whole blood transfusions tend to bring any abnormality in the chemical findings in the spinal fluid toward normalcy. Given repeatedly at intervals of one or two days, they tend to raise the fluid chlorides, carbonates, and *pH*. Even when bacteria are present, providing the lesion has not been too long established, these transfusions aid in healing a meningeal infection.

I have observed, later in the course of otogenic meningeal cases, when such transfusions have been employed, that there is less tendency to have "blocking" of the flow occur. These transfusions appear to keep obstructing factors from forming at the ventricular openings. The transfusions should always be small in amount: about 35 to 50 c.c. for children, and 75 to 150 c.c. for adults, varying according to the height, weight, and "largeness" of the individual patient.

The focal bone lesion is removed and in the face of an intracranial bacterial invasion, included in this procedure, I advocate exposing the visceral inner surface of the meninges upon which the infected temporal bone rests. By this I mean removing the entire inner table of the temporal bone, particularly all bone of the mastoid process, the entire tegmen cellulae and tegmen tympani, and the mesial wall of the petrosal pyramid. This is done to break the contiguity of the skeletal venous blood vessels, which penetrate these parts of the temporal bone, and are the usual route which the infection travels from the bone to the meninges. This done, the employment of spinal taps is continued.

Lately chemotherapy has been added to our resources. From the employment of sulfanilamide excellent results are now being reported. My own experiences with this drug have been very gratifying, and the results are the more brilliant, the truer and the surer is the complete removal of the antecedent bone focus. Sulfanilamide therapy and transfusions make an ideal therapeutic combination in treating otogenic meningitis. Because of its recent advent to our armentarium, it may not be

amiss to stress the manner of administration of this drug.

In using sulfanilamide by mouth, injection, or both, we are employing a bacteriostatic agent. I have recovered it in both the spinal fluid and in the blood in otogenic meningitis cases. There is no fixed dose for sulfanilamide. It must be given in sufficient quantities to procure results. There must be a concentration of the drug in both the blood and in the spinal fluid. My own experience tends to make me advise a daily examination of both blood and fluid during the period that the sulfanilamide is being administered to estimate its concentration in the body fluids. In addition, estimations of methemoglobin must also be made. This is necessary successfully to use the drug, as insufficient dosage is useless.

In children of average size and weight up to 10 years of age, I give 40 gr. daily at three hour intervals, by mouth, and in young infants, 10 to 20 gr., according to the severity of the infection. In adults the dosage is proportionately larger. The constant effort is to saturate the body fluids as rapidly as possible and to this end as much as 120 gr. have been given. When administered under strict control to meet its untoward effects, the dosage of the drug may cautiously be pushed even higher in some instances.

No other drug medication should be given with the administration of sulfanilamide. Sulphates are particularly to be avoided. Magnesium sulphate, commonly employed as a purgative, should be replaced by mineral oil or enemata. Because it replaces elements used up in the blood in the action of the chemotherapy when administering sulfanilamide, the combination of the drug with the employment of small whole blood transfusions is advantageous.

Finally, sulfanilamide should not be too soon or too suddenly withdrawn. The drug should be continued for at least a week after all symptoms have abated. During this week, the drug dosage should be lessened each day and so gradually stopped. For some time after a patient has been using the drug, exposure to high sunlight should be avoided. Skin rashes have been noted, and an intense itching of the skin has been complained about. In those in whom gastric symptoms appear, dosing with bicarbonate of soda is effective and alleviating.

During the administration of sulfanilamide, in the effort to produce a concentration of it in the body fluids, I have already alluded to the formation of methemoglobin. This may be produced to a degree that "air hunger" becomes noted, and cyanosis develops. Transfusions, when combined with the drug, tend to lessen this effect, yet it is a danger. Recently Professor William B. Wendel of the University of Tennessee has given us a means of combating the formation of methemoglobin. He recommends the intravenous injection of 0.1 c.c. of a 1 per cent solution of methylene blue per kilo of body weight for the purpose. This substance acts as a catalyzing agent, and releases the fixed oxygen element in the methemoglobin combination. The patient thus has more oxygen hemoglobin available for his needs. The use of this agent, in conjunction with sulfanilamide, has markedly lessened the signs of cyanosis, and also has permitted the employment of larger dosage with this drug.

From the above, it is apparent that the estimation of the methemoglobin is an important procedure during the treatment of otogenic meningitis, but this test has heretofore been a cumbersome one. In our hospital service a simplified colorimetric test with a minute amount of blood has been perfected and used. This is important, because the use of the veins at the elbow in taking a quantity of blood for methemoglobin estimation (employing the method of Marshall), is a handicap when these veins are needed for the repeated transfusions. They eventually thrombose and become obliterated.

A method devised by Dr. Edward A. Horowitz is therefore useful, because it obviates the necessity of employing venous

blood. The blood to be examined is taken from the finger tip; only 0.1 c.c. is required. Since the quantity of blood used is $\frac{1}{20}$ of that necessary in the Marshall technique, the quantity of each of the other reagents is reduced in proportion. The blood is drawn into a Byrd diluting chamber, the same as the one generally employed in laboratories for micro-Folin Wu determinations. The 0.1 c.c. of blood is drawn to the mark, then at once diluted with saponin drawn to the second mark (total volume 0.8 c.c.). After mixing, the diluted and laked blood is drained into a test tube and 0.2 c.c. of toluene sulfonic acid solution is added, with shaking. After five minutes the test tube is centrifuged. Most of the clear supernatant fluid is drawn into a Wright's pipette, and 0.4 c.c. of it is transferred to a special comparison tube, of the same length but only half the volume of an ordinary Sahli hemoglobinometer tube, graduated in the same way, to 140. The 0.4 c.c. of supernatant fluid reaches the 40 mark. Sodium nitrite, 0.04 c.c., is added, the mixture shaken, and allowed to stand three minutes for the dialization to become complete. Dimethyl-a-naphthylamine solution, 0.2 c.c., is now added, and the fluids fixed. The standard is prepared with 0.4 c.c. of a solution of sulfanilamide containing toluene sulfonic acid, treated like the blood specimen filtrate. The colors are compared by placing the tubes in an ordinary Sahli colorimeter, the solution of stronger color being diluted to match the weaker.

Horowitz states that the micro-sulfanilamide determination is not to be expected to be as accurate as the usual procedure, in which 2 c.c. of venous blood is used. However, he continues, it enables us to make blood sulfanilamide determinations with a frequency to which objection might be raised if venipuncture were required each time. In addition to the advantages in meningitis cases, where the veins are needed for other purposes, this method is believed to be useful in females in whom veins are reached with difficulty, and especially in children and infants.

The following case illustrates many of the points made in this paper regarding the diagnosis and the treatment of otogenic meningitis. The history is only briefly sketched, because the details are not germane to the argument here presented.

G. D., white male, aged 7, was admitted to my service at the Beth Israel Hospital, New York City, on April 17, 1938, giving a history of having undergone a bilateral mastoidectomy one year previously. Four months prior to admission there was an infection of the right middle ear and right mastoid process, which was evidenced by pain, redness and swelling over the process, and under the scar where the year old mastoidectomy had healed. The swelling eventually fluctuated and was incised, pus was evacuated and drainage instituted. Healing ensued. One month prior to admission following an attack of measles, the patient again complained of pain in the right ear. Paracentesis was performed. The following day swelling and fluctuation again appeared over the old scar which again was incised, the pus evacuated, and postauricular drainage established. The temperature ranged during this period between 101° and 102°F. I saw the patient during this period, examined him neurologically, and found him otherwise negative clinically.

Two days before admission, severe right sided frontal headache developed, and a right facial palsy became apparent.

On admission, the right mastoid showed an open postauricular wound from which a purulent pulsating discharge emanated. The middle ear was filled with a purulent pulsating discharge, coming away under evident pressure. Nose, throat and opposite ear were negative. Clinically the patient presented nuchal rigidity, bilateral Kernig, hyperesthesia of the right side of the face, sluggish corneal reflex on the right side. There was a distinct right facial palsy. The patient lateralized the tuning fork to the left (Weber). There was a horizontal nystagmus to the right.

On admission his blood study showed: hemoglobin 73 per cent; R.B.C. 4,300,000; W.B.C. 39,000, with polys, 88 per cent, monocytes 12 per cent, unsegmented staff cells 6 per cent. The spinal fluid gave 4,200 cells, with positive findings of Streptococcus hemolyticus from the first spinal tap taken. The roentgenograms of the mastoid process were essentially negative, but those of the petrous pyramid showed a degree of atrophy of the apico-carotid portion

of the temporal bone, on the right side. There was no dehiscence in the superior surface (petrosal ridge, using Stenver's position) of either pyramid.

The clinical picture of a meningitis developed very rapidly, so that from the clinicians' point of view the diagnosis was established. The spinal tap gave a pressure of 180 mm. of water; the fluid was turbid, contained 4,200 cells, 98 per cent of which were polynuclears, and 2 per cent lymphocytes. There was 26.7 mg. of carbohydrate, total protein 47 mg.; the blood chlorides were 490 mg., the spinal fluid chlorides 690 mg.; the blood carbonates were 42.8 mg., the spinal fluid carbonates 31.8 mg. The patient had been upon sulfanilamide therapy before coming under observation and therefore the lactic acid was not studied, since it was believed that this drug might influence the finding. The smear of the fluid was negative, but the culture gave the finding of Streptococcus hemolyticus.

The tentative diagnosis of otogenic meningitis was held to have been established, but I felt that, because of the lack of evidence of meningeal tissue reactions, the case was still in the phase of invasion rather than of infection. Blood transfusion was immediately given, 75 c.c. of whole blood being administered by the direct method; and sulfanilamide therapy was also started. The same day, an operative exploration of the right temporal bone was undertaken to seek and eradicate the focal bone lesion infecting the meninges.

The previously performed mastoidectomy wound was widely opened, its purulent contents and granulations eviscerated, and the entire inner table closely searched for a lead to a purulent focus. The basal portion of the petrosal pyramid was searched, and the osseous cellular tissue about the semicircular canals was probed for a fistula which might lead to the causative focal lesion. Posterior and superior to the labyrinth no such fistula was found. The inner table of the mastoid process was then removed and the tegmen cellulae also taken out. No extradural pus collection was found.

The tympanic cavity was then entered, converting the simple mastoidectomy into the procedure embraced in the performance of the radical. The postauricular bony wall was removed and the tympanic cavity opened, removing all the detritus, purulent granulations and the two major ossicles. Then, near the tubotympanic orifice, a large fistulous opening was found—anterior to the cochlea leading toward the petrous apex. This was enlarged until it was possible to enter it with a curette, and its purulent contents removed by curette and suction. A rather large rubber drain was introduced into this opening into the petrosal pyramid apex, after the removal of the tegmen tympani and some of the bone, necessary to expose the dura over the more forward lying parts of the petrous pyramid.

The surgical treatment is not germane to this discussion, and its subsequent details are omitted. Suffice for the purpose in hand to state that on the following day the spinal tap revealed only 690 cells in a cloudy fluid; 85 per cent of the cells were polynuclears, and 15 per cent were lymphocytes. On smear and on culture this fluid was sterile.

On the third postoperative day, the child began to improve clinically. In the interval, small transfusions and the sulfanilamide therapy had been continued. The neurologic signs disappeared, except for the facial palsy which, however, showed unmistakable signs of improvement. On the fifth postoperative day the spinal tap was clear, containing only 75 cells (30 per cent polys and 45 per cent lymphocytes). Carbohydrates had been regained to 43.1 mg. The total protein reaction was 36.8 mg. The culture and smear were negative. The facial palsy further improved, distinct movement in the region of the forehead being noted. The meningeal invasion was considered past, and a plastic closure of the superficial wound was undertaken. On May 8, 1938, the patient was discharged, normal except for a slight paresis of the muscles of the right side of the face.

The after treatment of the wound was carried out in the usual way, and the child is now completely recovered, the wound healed, the facial nerve also completely recovered. He was last examined by me during the last week of June 1938.

SUMMARY

1. In otogenic meningitis, the particular bacteria invading the central nervous system play in themselves a relatively small rôle; the tissue reactions and the clinical signs are almost similar, irrespective of the nature and kind of invading microorganism.

2. Clinical evidence stresses the fact that the symptomatology is divisible into

two groups of clinical signs: those due to increased intracranial pressure, and those due to toxicity, including the terminal sepsis from bacterial activity. Where the predominating symptoms are those of pressure, if this can be relieved, cure will not necessarily follow unless the other factors incidental to the lesion have been mastered. If the intracranial pressure is abated, time is gained to carry on measures to combat the other factors. If the intracranial pressure remains high and mounts, then death intervenes before the other factors in the case can be handled.

3. Drainage of the central nervous system does not answer the problem presented in meningitis. The problem is more in keeping the brain tissue alive to outlast the infection. The important step to accomplish this is to keep the cerebrospinal fluid circulating. Any procedure or any technique which stops this circulation defeats one of the objects in view. Brain cells naturally function best, and their by-products of cerebral cell metabolism are most easily neutralized, when they are kept bathed in a cerebrospinal fluid, the chemistry of which is as near normal as possible. The deposit of plastic exudate, which impedes cerebrospinal circulation, is to be lessened to the greatest possible degree. This is best accomplished by the administration of small whole blood direct transfusions, which are begun as early as possible in the course of the disease and continued until all meningeal symptoms have disappeared.

4. Chemotherapy, in the form of sulfanilamide, has demonstrated distinct value. It may be given by mouth, injected intramuscularly, or in a combination of both means. During its administration, repeated tests for methemoglobin should be taken. Combined with the blood transfusions, the dangers from the reactions to the drug are lessened.

5. No evidence is as yet at hand that medical treatment alone suffices to give a reasonable assurance of obtaining consecutive recoveries. It is therefore absolutely necessary that every possible bone focus be thoroughly removed as soon as its presence is diagnosed. Additionally, the breaking of the contiguity of structure of the skeletal dural veins—a frequent route for meningeal invasion—gives another factor toward obtaining recovery. In diagnosis, the clinical picture alone is insufficient to furnish the clinician with specific diagnostic data. In general terms, it may suffice to establish a clinical diagnosis of otogenic meningitis, but since an exact determination of the phase which the given case presents is highly desirable—so as to institute intelligent remedial therapy—data which furnish the basis for this determination must be sought. Clues are to be found in repeated spinal tap examinations, and in comparisons of the fluid contents with those of the circulating blood. In fluid examination, its physical properties, the pressure under which it is obtained, its cytology, its bacteriology, and lastly its chemistry are exceedingly important.

6. With these newer conceptions of otogenic meningitis, more recoveries are being reported, and we meet meningeal infections with more confidence because we are better able to combat them. With this hopeful note on the future additional developments in therapy, let me, for the present, leave the problems presented by otogenic meningitis.

REFERENCES

Kopetzky, Samuel J. Meningitis, nature, cause, diagnosis, and principles of surgical relief. An experimental and critical study. *Tr. Am. Laryng., Rhinol., and Otol. Soc.*, 1912.

Kopetzky, Samuel J. Notes on the diagnosis of otitic meningitis. *Ann. Otol., Rhin. & Laryng.*, 43: 401, 1934.

Kopetzky, Samuel J., and Fishberg, E. H. Early diagnosis in meningitis; chemical studies of the spinal fluid. *Arch. Otolaryng.*, 16: 851, 1932.

Kopetzky, Samuel J. Management and treatment of otogenic meningitis. (Presidential Address.) *Ann. Otol., Rhin. & Laryng.*, 47: 117, 1938.

Wendel, William B. Lecture delivered before Midwestern Section, Am. Laryng., Rhin. & Otol. Soc., St. Louis, Mo., February 1938. In press.

Horowitz, Edward A. Hyperpyrexia or sulfanilamide in the treatment of gonorrhea in women. *M. Clin. North America*, in press.

THE RADICAL MASTOID OPERATION

JOHN MACKENZIE BROWN, M.D.

Clinical Professor of Otolaryngology, University of Southern California School of Medicine

LOS ANGELES, CALIFORNIA

DURING the ten years following the inception of the radical mastoid operation by Stacke and Jänsen in 1889 the procedure was acclaimed with almost wild enthusiasm, bringing about a large number of poor results in patients who would have been better off without surgery. Since this period, otologists throughout the world have, in general, swung to a much more conservative stand. That this stand may be even too conservative must now be considered. Courville and Nielsen have shown in a survey of 10,000 consecutive autopsies that there was an incidence of middle ear infection amounting to 1.67 per cent; Kafka has shown that out of 1,125 cases of chronic mastoiditis, 7 per cent developed intracranial complications and of these, 87 per cent terminated fatally. Therefore one might say that surgery in cases which have already developed intracranial complications is almost tantamount to locking the barn after the horse is stolen.

The primary reason for doing the radical operation is to safeguard the patient's life. In the absence of imminent dire complications this operation may be considered for the elimination of chronic, foul-smelling discharge, troublesome to the patient either socially or economically in that it may interfere with his getting employment. The preservation of the remaining hearing in these ears is desirable but should not be stressed too strongly if other and more important reasons for doing the operation are present. As Ballance has said, "It is evident that there is still undue hesitation in advocating operation before the onset of threatening symptoms. The disease itself is the indication for operation, not the threatening symptoms of wider and deeper disease. The most truly 'conservative' treatment is that which most surely and speedily cures the disease."

The indications for doing the radical operation may be divided into two major groups. The first group comprises the imperative indications, such as intracranial complications. Meningitis in the course of a chronic otitis media is certainly an indication for operation. If operation is done early during the period of threatened meningitis (serous meningitis or meningismus) the chances of a satisfactory termination are much greater than when frank meningitis exists. This latter complication is nearly always fatal.

When lateral sinus thrombosis occurs in chronic otitis media, experience shows that there is widespread bone destruction which in most cases involves the tegmen antri or the tegmen tympani as well. The operation here is a safeguard from further extension, possibly to the brain, as well as for the thrombophlebitis itself. Brain abscess, if present, almost always occurs through direct extension. Therefore, the most logical approach for surgical relief is by means of the radical operation during which the fistulous tract will be uncovered. Should a basal skull fracture occur, involving the middle or inner ear, in a patient suffering with chronic otitis media, immediate radical surgery is indicated if the patient's condition warrants it, as such a fracture lays open the way for meningitis or purulent labyrinthitis.

When labyrinthine irritation occurs repeatedly during the course of chronic otitis media it usually means necrosis involving the labyrinthine capsule and most often at the horizontal canal. Either this or the demonstration of the presence of a labyrinthine fistula is an imperative indication for radical surgery to avoid the possible

onset of labyrinthitis. However, when labyrinthitis does occur a more conservative stand is advocated. This viewpoint is summed up by Coates, who says, "Not all labyrinthitides of stormy onset are purulent. A diffuse serous labyrinthitis may start suddenly with almost identical symptoms, but will recover even with retention of more or less function. To operate on such a case would be a mistake. Furthermore, in those cases of acute suppuration of the labyrinth that do not follow traumatic introduction of infection, many surgeons prefer to delay operation until the violent acute symptoms have subsided and the quiescent period has been inaugurated, unless there are present the earliest signs of beginning intracranial involvement, in which case immediate operation is imperative." Friesner makes the observation that when labyrinthitis occurs during the course of chronic otitis media, it is more innocuous than that occurring either during the course of an acute otitis media or mastoiditis or that following a traumatic injury to the labyrinth.

Although it is possible to conceive of a Bell's palsy occurring during the course of a chronic otitis media, such cases must necessarily be rare. The usual cause is the involvement of the nerve, by extension of the primary lesion, somewhere in its course through the Fallopian canal. Few, if any, otologists would assume the responsibility of not operating in such a case immediately if the patient's condition permits.

Neoplasms involving the middle ear, mastoid process, or external canal are indications for immediate radical surgery followed by irradiation. Schall states that this condition is not so rare as formerly thought. The most constant finding is otorrhea and the most suggestive symptom a tendency to bleeding. Although pain may be early, it does not usually occur until the growth causes pressure. Facial paralysis is comparatively rare. Aural polyps which bleed excessively on removal are usually the earliest sign and it is therefore recommended that microscopic sections should be made of all aural polyps removed. Roentgenograms do not show the early stage.

Cholesteatoma at times may be an imperative indication for surgery. It is best discussed under the relative indications which form the second or larger group. It is in this group that the surgeon's judgment and experience are most important. Cholesteatoma has been widely discussed and extensively studied since it was first given its name by Mueller in 1838. It has been classified as primary and secondary, wet and dry, but from the standpoint of this paper it will be discussed only as to the indications for or against surgery when it is present.

Ruttin describes five types of perforations of the tympanic membrane with the treatment indicated for each type. In the first type there is involvement of the eustachian orifice and although discharge may be continuous there are rarely any complications. The radical operation is not indicated and, if done, a dry ear is not to be expected. In these the perforation is anterior. The second type is chronic otitis media with a central or kidney-shaped perforation which may be quite large but without involvement of the tympanic ring. In these the discharge is mucoid, and granulation tissue or polyps may be present. In general the hearing is good and there is no bone necrosis or cholesteatoma. This type responds well to conservative treatment, seldom has complications and the radical operation, if done, is rarely successful. The third and fourth types comprise those perforations of Shrapnell's membrane anteriorly or posteriorly which involve the lateral attic wall. Complications, especially cholesteatoma, are frequent and some type of radical operation should be done. His fifth type is that in which there is a small central perforation. This type does not lend itself to the conservative operation. Rüedi states that cholesteatomas with large perforations are more prone to give suppurative mastoiditis

and facial paralysis, whereas small perforations in the presence of a cholesteatomatous process are more apt to give intracranial complications, such as extradural abscess, meningitis, and brain abscess. It is therefore possible to predict somewhat the course which cholesteatomatous complications are likely to run. Rüedi also states that the cholesteatomas with small perforations must be treated by otologists in an entirely different way, as they do not lend themselves to conservative treatment, are potentially dangerous to life, and are predestined to radical operation in order to avoid complications.

In cholesteatoma unaccompanied by secondary infection, that is, of the "dry" type, conservative measures are more apt to meet with success than in the "wet" type. As all cholesteatomas are markedly hygroscopic, the patient must be warned not to get water in his ear. He should be cautioned to wear a tight water proof cap over his ears or tight fitting plugs if he wishes to swim. The attic cannula may be used to irrigate these cavities. The cholesteatomatous material may be softened with peroxide, but alcoholic solutions should be used afterwards. Ninety-five per cent alcohol is recommended by many authorities and Rejtö states that no strength less than absolute alcohol should be used. He goes even further to recommend the use of carbon tetrachloride which is a marked fat solvent.

The result of the conservative treatment of the purulent or "wet" type of cholesteatoma is not nearly so satisfactory. When chronic otitis media, in which cholesteatoma is found or is strongly suspected, does not yield to a reasonable amount of thorough conservative treatment, the radical mastoid operation is indicated, as it is in this type that most intracranial complications occur. In this connection it might be said that although the complications of a chronic otitis media are generally associated with perforations of Shrapnell's membrane or in the attic region, and that chronic middle ear infections with central perforations are considered to be safe as far as complications are concerned, Zeisner states that Uffendorfe has reported two and Englehart one case, and he himself reports three of his own, in which large central perforations were present and which subsequently developed intracranial complications.

When surgery is indicated in a tuberculous otitis media nothing less than the total mastoidectomy should be considered. Tuberculosis of the middle ear is practically always secondary to lesions elsewhere. Guthrie states that tuberculous otitis media in children offers a grave prognosis. In all of his cases the disease commenced in the first year of life and was definitely traceable to milk infection. He, as well as Graham, Muskat and Scobee, believes that tuberculous otitis media is a much more common lesion than is generally recognized, and because of its insidious, usually painless, onset has not received proper attention. Scobee stresses the importance of a routine examination for the tubercle bacillus in children whose ears show a chronic discharge. He also states that tuberculous mastoiditis may have all or none of the characteristics of mastoiditis due to other organisms. Tuberculous ears present an added focus to the primary pulmonary lesion and do much to prevent its healing. While there is a definite risk of causing an acute exacerbation of the chest condition, Muskat infers from his own series that patients with active pulmonary disease improve in health following radical mastoidectomy. The operation should be done under local anesthesia if possible.

Should an acute otitis media develop after a basal skull fracture involving the middle or inner ear a radical operation may be necessary to provide an area of satisfactory drainage to the outside so that inward extension will not occur. The development of an acute petrositis in the course of a chronic middle ear infection usually indicates a radical mastoidectomy. If an acute petrositis develops during the course of an acute mastoiditis it may be

necessary to perform an extensive total radical operation in order to reach the fistulous tract should it be in the anterior portion of the middle ear. Fistulous openings into the cortex of the mastoid bone or in the bony auditory canal, often found in patients who have had previous simple mastoidectomies, may require a radical operation.

When imperative indications for the radical mastoid operation arise there is no alternative. However, it is a serious procedure, and it must therefore be stressed that it should be used for a definite purpose to accomplish a result important to the patient, either from the standpoint of his personal safety or his economic or social welfare. It should not be undertaken, in the absence of imperative indications, until the most intelligent, careful and painstaking conservative treatment has failed. This should include frequent personal observations and treatment by the otologist himself as well as careful supervision over the patient's home treatment. The removal of polyps and granulations to insure better drainage, the washing out of cholesteatoma when possible with the attic cannula, the use of alcoholic and astringent solutions and boric and iodine powders—these are necessary measures. At this point it may be well, also, to stress the careful examination and recording of the patient's hearing level as a routine procedure from time to time, which might even be of some medicolegal importance following the radical operation, should it be done later, as has been brought out by Page.

In children the radical mastoidectomy for the cure of chronic discharge alone is never justified. Here the definite indications for the radical operation are intracranial complications, cholesteatoma and tuberculous otitis media. Guthrie states that cholesteatomas, polyps and caries of the ossicles are rare under the age of ten. Page offers statistics to prove that the increased number of tonsillectomies and adenoidectomies in the past eighteen years have materially cut down the number of cases of chronic otitis media. He gives three rules for its prevention: (1) tonsillectomy and adenoidectomy in children with repeated upper respiratory infections; (2) early myringotomy in recurrent otitis media; and (3) simple mastoidectomy at the first sign of widening of the perforation in the drum head.

Adequate, intelligent, conservative treatment of chronic otitis media in children will yield a high percentage of cures. Asherson states that 50 per cent of children have central perforations. It is in this type that tonsillectomy, adenoidectomy and removal of nasal foci is of great importance. Forty per cent of the remaining cases of chronic otitis media in children have marginal perforations with granulations and local bone disease. When actual bone necrosis is present, nose and throat surgery does not influence the course of the disease.

Mygind, among others, stresses the complete medical examination of these children, including hemoglobin, Wassermann, Pirquet test, and determination of the presence of rickets, as well as the taking of the family history as to ear trouble, catarrh, and tuberculosis, the home environment and diet. He also mentions a type of child predisposed to chronic ear infection. These predisposing factors which Wittmaack has described in the middle ear and mastoid are, he believes, also at work throughout the body, and may be evidenced by such manifestations as a disturbed water metabolism. He describes one type patient as asthenic and tuberculous, and more frequently, the obese, congested, "spongy" type of individual with cold hands and cyanotic extremities. In these cases rickets is not uncommon.

When definitely indicated, the complete radical mastoidectomy should be done. If hearing is at stake and especially if a bilateral operation is to be undertaken in children who have not yet learned to talk, some modified type of operation may be considered if feasible. However, when a substitute radical operation is done, these cases require more after care and closer

observation for recurrence or extension of the disease.

The results of the radical mastoid operation in children are disappointing according to Guthrie, who reports only 25 per cent cures in his cases. It has been the author's experience at the Children's Hospital in Los Angeles that the results are comparatively good as to dry ears, although good results are not so common as in adults.

Prevention of chronic otitis media should be stressed as well as hygiene and conservative treatment. The radical mastoidectomy in children should be done only in the presence of definite, urgent indications or as a measure of safety when all other treatment fails, excepting in those cases in which the discharge is from the mucosa only and evidence of tuberculosis or cholesteatoma is absent.

That the valuable results of the radical mastoid operation are obtained by sacrificing some or all of the remaining hearing seems to be the consensus of opinion. Some men believe with Pressman that although theoretically hearing should be improved by the removal of the thick functionless tympanic membrane, the partially destroyed ossicles, polyps and granulation tissue (which all tend to obstruct hearing in the middle ear and which may serve as a focus of toxic absorption causing further labyrinthine degeneration), actually the majority of cases, while not improved, at least have no further hearing loss. Kopetsky states that in chronic otitis media of any great duration the hearing deteriorates in operated or unoperated cases, and therefore it is better to operate early with the hope of obtaining a dry ear though loss ensues, than to operate years later in the presence of a dangerous lesion when it will be found that actually the hearing loss will be as great or greater than had the ear been operated earlier.

When there is no alternative the hearing will, of course, not be considered. When both ears are involved or in those cases where surgery is indicated but the hearing is good, and especially in children, young adults, or those whose very livelihood depends upon what hearing they have left, the question of preservation of hearing must be seriously considered. It is because of this unsatisfactory phase of the total radical operation that so many types of incomplete or partial radical operation have been developed. Among these may be mentioned ossiculectomy, which by now seems to have been almost entirely abandoned; various types of intrameatal attico-antrotomies; the conservative radical operation in which the bony posterior meatal wall is left intact, the antrum opened widely and the incus removed if diseased; the modified radical operation or attico-antrotomy in which the posterior bony canal wall is lowered and the bridge removed as well as the lateral attic wall, and the cholesteatoma gently curetted out, great care being taken not to disturb the ossicular chain. Some operators remove the incus if it is diseased.

Of these operations the last, or attico-antrotomy, developed by Bondy, has been used with the most success as a substitute for the total radical operation in carefully selected cases, that is, those with a marginal attic perforation and the presence of cholesteatoma, and in the absence of imperative indications for the total radical mastoidectomy. It can later be converted into a total radical operation through the meatus under local anesthesia should this procedure become necessary. This operation is done for the conservation of hearing and if hearing is not present or at such a low level as to be almost valueless, the remaining ear having good hearing, there is obviously no advantage in this procedure over the total radical mastoidectomy.

The after care in attico-antrotomies is much more difficult and requires more attention than in the total operation. The recurrence of cholesteatoma must always be held in mind. The necessity for strict supervision and observation in these cases is brought out by Howarth and Bateman. In a series of eighty-three cases in which

the conservative operation was done, 20 per cent were reoperated for persistent discharge and alarming symptoms. These usually showed a large suppurating cavity in the mastoid, high up in the region of the tegmen antri and the roof of the aditus, usually with much exposure of the dura which was thickened and covered with granulations. Some of these reoperated cases had been dry for a variable period of time before suppuration reoccurred. Of three cases in which this operation was done by Bárány himself the author later had to reoperate in all because of recurrent cholesteatoma. With the advent of perfected hearing aids the question of preservation of the remaining hearing should influence the surgeon less and less when surgery is indicated.

The total radical mastoid operation is now classical, but there are various points which may well be brought out in a description of this procedure. That failure to achieve satisfactory results does occur when most painstaking surgery has been done by a skilled operator is unfortunately true. Many postoperative failures, however, are due to the lack of scrupulously adhering to a certain rigid technique in the performance of the operation. If the surgeon develops a certain invariable routine he not only improves his own skill by constant repetition, but, by following a set course of procedure, is not apt to overlook any area requiring surgical attention.

The usual incision is made a quarter of an inch behind the fold of the ear and parallel to it, from the zygomatic region to the tip of the mastoid process, and is carried through the periosteum to the bone. Kully's incision starts in the scalp one-quarter inch posterior to the hairline and is carried forward into the anterior root of the zygoma on a level with the upper border of the external ear. The incision is extended downward paralleling the curve of the external ear, then forward to the lower border of the tip. The periosteum is then elevated forward to the posterior root of the zygoma, posterior canal wall and tip of the mastoid, and held with a hand retractor. The advantages are the same as those above plus a richer blood supply and no visible scar except for one-half inch at the tip.

Finding the antrum and aditus is often difficult because of sclerosis which may sometimes even obliterate the antrum and greatly narrow the aditus. The lateral sinus may lie far forward in the antral region or the bony canal may be oblique. In such cases the safe way to find the aditus is to follow down the posterior superior canal wall, even exposing the dura if necessary. In this way the horizontal canal and facial nerve are avoided. The mastoid cavity should be cleared of all infected areas, but there is no advantage in removing healed sclerotic bone from the mastoid process if the infection is localized to the area surrounding the antrum.

The posterior canal wall, having been brought down to the bridge, the latter may now be bitten through with an aditus punch or a small curette may be placed on its under side, the cutting edge held toward the operator and parallel with the tegmen antri and rotated from side to side. Great care must be taken on removing the bridge, not to injure the facial nerve where it makes its last bend.

The remains of the malleus, incus and tympanic membrane are now removed. The external wall of the attic and the upper part of the bridge are removed to the level of the tegmen tympani. The hypotympanic space is obliterated by removal of the annulus and lowering of the floor of the bony meatus to give a downward and outward slope. The anterior wall of the middle ear should be thinned and the annulus removed. The mucous membrane of the eustachian orifice should be thoroughly curetted. The close proximity of the carotid artery in this region must be kept in mind as well as the fact that dehiscences do occur. The floor plate of the canal for the tensor tympani should be opened and the tendon removed. Great care must be taken in the removal of granulations and polyps

from the region of the promontory. The region of the oval window should not be disturbed, also the facial nerve as it runs above the oval window to its second bend lies beneath a very thin plate of bone and can often actually be seen through it.

The facial ridge should now be lowered to the limit of safety with a small chisel or Richards' curette paralleling the nerve so that if it should be inadvertently exposed, further injury may be avoided. The stylomastoid artery, if encountered, indicates that the facial nerve is dangerously near. As much as is safely possible should be removed of the overhang where the facial ridge forms the posterior wall of the middle ear. One should now inspect the operated area, smoothing all rough surfaces.

The plastic operation on the auricle and membranous meatus is very important as it is by this procedure that the operative cavity is partially lined by skin which is to aid in its epithelization. Poorly made flaps are one reason for unsatisfactory postoperative results as it is necessary to have adequate exposure of the radical cavity to allow a direct visualization of all areas during the period of after care.

A large hemostat is inserted into the external auditory meatus and an incision made from below upward to the bowl of the concha. Incisions are made upward and downward from this wound so that the result is a "T." The resulting quadrilateral flaps are carefully trimmed of their underlying cartilage and are molded into the mastoid cavity where they are retained by twenty day chromic catgut sutures taken through the subcutaneous tissues of the posterior wound and left buried. A half inch strip of iodoform gauze is now introduced through the meatus and the entire cavity is packed firmly but not tightly, for if it is packed too tightly there is interference with drainage and the growth of normal granulation tissue. The periosteum is closed tightly with interrupted stitches of twenty day chromic, the skin incision may be closed with skin clips or dermal sutures as the operator desires. If there has been much involvement of the tip, a small drain may be inserted at the lower end of the wound, to be removed in four or five days.

Various types of primary and secondary skin grafts, muscle and periosteal pedicle flaps, fat transplants, and even amniotic membrane have been used for the purpose of lining or obliterating the mastoid cavity. It has been the author's experience that very satisfactory results are obtained without these procedures. However, it is stated by those who use them that faster healing can be expected.

The final success of a radical mastoidectomy can be said to depend on the after care. Here it is imperative that the surgeon do the dressings himself. The first dressing should be done on about the fifth day, at which time the skin clips or dermal sutures may be removed, the iodoform gauze pack removed and the cavity swabbed dry with sterile cotton. A strip of plain one-half inch gauze of which the tip has been covered with vaseline, is now introduced and packed firmly into all parts of the cavity as before. If packed too tightly there is interference with normal epithelization, and if packed too lightly unwelcome proliferation of granulations may occur. The vaseline on the tip insures less bleeding and less discomfort to the patient when the packing is removed. The patient should be seen every day but the packs are changed every day to every other day depending upon the condition of the wound. The average patient should be able to leave the hospital at the end of one week and further dressings may be done in the office. Every five to six days the pack should be left out for twenty-four hours and then replaced. The packs are changed every other day, except for the periods of aeration, for about four weeks, at which time the cavity should be well epithelized. Excessive granulations may be touched up with a small bead of 75 per cent silver nitrate on the end of a wooden applicator. The stick is

preferable to the use of solutions which may spread and destroy new areas of epithelization. After the wound is completely healed the patient should be seen every three months for the first year at which time audiometric tests should be recorded. Subsequently the patient should be seen at least twice a year.

SUMMARY AND CONCLUSIONS

When imperative indications for the radical mastoidectomy exist there seems to be no difference of opinion as to the advisability of immediate operation.

When relative indications exist, the judgment and experience of the operator is most important in the selection of cases which may be benefited by radical surgery.

At the present time the tendency is toward ultra-conservatism so that patients who would be greatly benefitted by this procedure are carried on under conservative treatment for long periods of time until imperative indications may arise.

The perfection of modern hearing aids should do much to encourage earlier operation when actually indicated.

In the absence of imperative indications, comprehensive conservative treatment should at first be carried out together with thorough study of the individual case, not only as to his ear condition but as to his general physical condition; history; family, social and economic background.

The condition of the labyrinth should be ascertained prior to surgery.

The hearing level should be studied and recorded audiometrically several times before surgery is undertaken.

The final success of the radical mastoidectomy depends on the skill of the operator and minute care as to detail, as well as painstaking after care.

REFERENCES

ASHERSON, N. Some postoperative results of the radical mastoid operation in children. *J. Laryng. & Otol.*, 47: 317–332, 1932.

BALLANCE, SIR CHARLES A. Essays on the Surgery of the Temporal Bone. London, 1919. MacMillan.

BONDY, G. Totalaufmeisselung mit Erhaltung von Trommelfell und Gehörknöchelchen. *Monatschr. f. Obrenh.*, 44: 15–23, 1910.

COATES, G. M. Discussion of some recent developments in regard to otosclerosis, cholesteatoma and statis labyrinth. *Laryngoscope*, 43: 622–629, 1933.

COURVILLE, C. B. and NIELSEN, J. M. Intracranial complications of otitis media and mastoiditis. Statistical study with survey of 10,000 necropsies. *Acta oto-laryng.*, 21: 19–60, 1934.

FRIESNER, I., and ROSENWASSER, H. The treatment of labyrinthitis. *Arch. Otolaryng.*, 20: 139–151, 1934.

GRAHAM, H. B. Taking stock of the radical mastoid operation. *Tr. Am. Laryng., Rhin. & Otol. Soc.*, 37: 441–447, 1931.

GUTHRIE, D. The prognosis of middle ear suppuration in children. *J. Laryng. & Otol.*, 41: 305–315, 1926.

GUTHRIE, D. Mastoid surgery in childhood. A record of fifty consecutive operations, with a note on the value of fat-grafting. *J. Laryng. & Otol.*, 43: 713–723, 1928.

HOWARTH, W., and BATEMAN, G. Mastoid operations. *Brit. M. J.*, 2: 954–955.

KAFKA, M. M. Mortality of mastoiditis and cerebral complications with review of 3,225 cases of mastoiditis with complications. *Laryngoscope*, 45: 790–822, 1935.

KOPETZKY, S. J. Acute and chronic otitis media and sinus thrombosis; summary of bibliographic material available in field of otolaryngology. *Arch. Otolaryng.*, 14: 489–511, 1931.

KOPETZKY, S. J. Cholesteatoma. *Laryngoscope*, 43: 118–131, 1933.

KOPETZKY, S. J. Acute and chronic otitis media, sinus thrombosis and petrosal pyramid suppuration; summary of bibliographic material available in field of otolaryngology. *Arch. Otolaryng.*, 20: 396–421. 1934.

KOPETZKY, S. J. Acute and chronic otitis media, sinus thrombosis and suppuration of petrous pyramid; summary of bibliographic material in field of otolaryngology. *Arch. Otolaryng.*, 22: 336–357, 1935.

KOPETZKY, S. J. Acute and chronic otitis media, sinus thrombosis and suppuration of petrous pyramid; summary of bibliographic material available in the field of otolaryngology. *Arch. Otolaryng.*, 24: 505–526, 1936.

KOPETZKY, S. J. Diseases of petrous portion of temporal bone from standpoint of general medical man, neurologist and otologist. *J. Iowa M. Soc.*, 26: 451–455, 1936.

KOPETZKY, S. J. Chronic mastoiditis and its therapy. *J. Iowa M. Soc.*, 26: 665–670, 1936.

KULLY, H. E. Periosteal flap in performance of mastoidectomy. *Laryngoscope*, 46: 537–540, 1936.

MUSKAT, I. Eradication of chronic tuberculous middle ear and mastoid disease in tuberculous patient. *Am. Rev. Tuberc.*, 28: 447–452, 1933.

MUSKAT, I. Radical mastoidectomy in adults with aural tuberculosis and active pulmonary tuberculosis. *Arch. Otolaryng.*, 22: 143–153, 1935.

MYGIND, S. H. The indications for radical operation in chronic middle ear suppuration. *J. Laryng. & Otol.*, 47: 297–316, 1932.

Pressman, J. J. Residual hearing after radical mastoidectomy. *Laryngoscope*, 41: 808–820, 1931.

Rejtö, A. Ueber die Behandlung der cholesteatomatösen Mittelohreiterungen. *Acta oto-laryng.*, 20: 230–234, 1934.

Rejtö, A. Ueber die Behandlung cholesteatomtösen Mittelohreiterungen. *Monatscbr. f. Obrenb.*, 69: 544–548, 1935.

Ruedi, L. Zur Klinik und Therapie des Mittelohrcholesteatoms. *Schweiz. med. Wchnscbr.*, 64: 411–418, 1934.

Ruttin, E. Zur typischen und konservativen Radikaloperation des Mittelohrs. *Monatscbr. f. Obrenb.*, 70: 1217–1229, 1936.

Schall, L. A. Neoplasms involving middle ear. *Arcb. Otolaryng.*, 22: 548–553, 1935.

Scobee, J. E. Tuberculous mastoiditis in infants and in children. *Arcb. Otolaryng.*, 17: 328–329, 1933.

Wittmaack, K. Cholesteatombildung Pneumatisation und Pneumatisationslehre. *Arcb. f. Obren-, Nasen-, u. Kablkopfb.*, 125: 218–234, 1930.

Zeisner, C. Komplikationen bei zentraler Perforation. *Ztscbr. f. Laryng. Rbin.*, 20: 28–30, 1930.

Every doctor is, or should be, aware of the fact that information obtained from a patient which is necessary in diagnosis or treatment is confidential . . . While the laws governing privileged information vary in different localities, the general legislative tendency has been to protect the public interest, safety, and health.

From—"The Roentgenologist in Court" by Samuel Wright Donaldson (Thomas).

THE EDUCATION OF THE DEAF AND HARD-OF-HEARING CHILD

MAX A. GOLDSTEIN, M.D., LL.D.

Director, Central Institute for the Deaf

ST. LOUIS, MISSOURI

BEFORE discussing the education of the deaf child, it might be well to classify briefly the different types and degrees of deafness found in child life.

CONGENITAL DEAFNESS

Congenital Deafness, or, as it has been erroneously labeled for many past decades, "deafmutism," is that form of deafness resulting from the transmission at birth of a physical imperfection or a pathologic process affecting the otitic capsule or brain in its embryologic development, or some histopathologic element of or in the peripheral or central areas associated with the organ of hearing. Congenital deafness, then, may be (*a*) biologic; (*b*) pathologic.

(*a*) *Biologic Congenital Deafness.* This must have as its basis the development of an imperfect embryo, in the growth formation of the otic capsule, temporal bone, brain or developed histologic structures associated with the mechanism of hearing. Thus, there may be absence or abnormality of essential structures of the temporal bone; a deformed cochlea; histologic deformities of the organ of Corti; presence of a nerve sheath but absence of a neuroglia of the cochlear nerve; undevelopment or irregularity of that portion of the brain cortex in the temporal area associated with the hearing centers. These may be enumerated among histologic or osteologic defects legitimately classified as etiologic factors of *biologic congenital deafness.*

(*b*) *Pathologic Congenital Deafness.* This includes the transmission of hereditary tendencies to deafness, through consanguinity in marriage, syphilis, tuberculosis, rickets, intra-uterine hemorrhage, etc.

This classification and discrimination of two types of congenital deafness is offered by the writer not only as an etiologic differentiation, but also for its possible prognostic value in the application of certain forms of pedagogy.

Another fact of significance and value for pedagogic purposes is that *over 30 per cent of reported congenitally deaf cases exhibit a residuum of hearing amenable to reëducation and stimulation.* Often this stimulation is of sufficient value to recover hearing in a given case that may make it practically possible for such a subject to receive much of his training directly through the ear. This valuable asset will be discussed in a later paragraph on education of the deaf.

Other terms similarly misapplied and having no scientific value are: semi-deaf and semi-mute, applied to those who have some slight power of hearing or some power of speech, either because the hearing is not totally absent or because deafness occurred after speech had been acquired. These terms semi-deaf and semi-mute have never had an excuse for being. They are inaccurate because there is no consistent scientific measurement or standard of such deafness; they confuse and complicate literature and should be barred from the nomenclature of this subject.

Residual hearing is found in about 30 per cent of all congenitally deaf children. The degree of hearing may vary from a bare appreciation of loud voice to a capacity for differentiation of pitch and vowel sounds and even some consonants; or, it may even include the hearing of familiar words and short phrases. This would seem to indicate that some part of the cochlear mechanism is still functioning.

The presence of residual hearing can be determined by tests with the voice at close range, and when this is found the child should be given the opportunity of training by what is generally termed the acoustic method.

The acoustic method, as defined by me some years ago, is "*Stimulation or education of the hearing mechanism and its associated sense organs by sound vibration as applied either by voice or any sonorous instrument.* This definition is comprehensive enough to include:

(*a*) Voice and musical sounds directed through the physiologic tract of the ear either to the peripheral or central auditory areas.

(*b*) Sound vibration as sensed by tactile impression to interpret pitch, rhythm, accent, volume.

(*c*) Analysis of speech sounds by tactile differentiation.

(*d*) Synthesis and speech construction by tactile impression.

(*e*) Sound waves and their significance as appreciated by optical perception.

An additional value of the inclusive expression "acoustic method," is to clear the atmosphere of a confusion of such terms as "auricular training," "aural gymnastics," "aural method," and to embody all recent research, such as radio and telephone principles, interrelation of the special sense organs, and a wider appreciation of the physiology of hearing.

Many a child with a considerable residuum of hearing has been found enrolled in state and even in some day schools for the deaf, to whom the privilege of training by the acoustic method and salvage of this residual hearing is not accorded. It may also be observed that there are congenitally deaf pupils with a considerable amount of hearing who are in schools for the deaf and are still being taught by the "sign method." Under these circumstances and in the light of modern pedagogy for the deaf, the training of such a child by signs must be condemned.

There is another phase of congenital deafness that has not been generally recognized, namely, "word deafness." In this type, the child may hear what is being said, but is unable to analyze and interpret the sounds heard. If placed in the right pedagogic groove such children frequently make remarkable progress in a comparatively short period and can carry on later in the normal schoolroom.

Another type of congenital defect that comes to the attention of the otologist and the teacher of the deaf is that known as "congenital aphasia," or the mechanical inability to produce speech, dependent on retarded development of the speech center or associated fibers, and often found in children with perfectly normal hearing. These cases are frequently incorrectly diagnosed as congenitally deaf, when the factor involved in such a child's physical defect is not one of hearing but of speech deficiency.

Congenital "word deafness" is the term used when there is an inability to understand and to use spoken language and may exist in the presence of normal physiologic hearing for the entire range of tone frequencies as tested by the audiometer. In congenital aphasia the speech fields are involved and may be dependent on an undeveloped speech-hearing center (Wernicke) or speech-producing center (Broca) even though the connecting tracts may be intact; or both speech-hearing (Wernicke) and speech-producing (Broca) centers may be intact and the associated tracts defective.

Differentiation of these somewhat similar and unusual types of speech defects which are apparently hearing deficiencies, is difficult, and in some instances these final differentiations cannot be made until the case has been carefully observed for a period of weeks or months. Sometimes there is difficulty in differentiating between a child having a language disability due to involvement of the cortical speech areas, on the one hand, and a congenitally deaf child, on the other.

HEARING TESTS

In the case of the young child we are frequently confronted with the question of the parents. "Doctor, is my child deaf?" To answer this question intelligently and conclusively is not always an easy task. There are but few tests of hearing that can be accurately applied in the case of a child under two to three years of age. At this age even the normal boy or girl has not acquired sufficient knowledge of speech to insure correct response to questions put to him with voice. The voice response in subjects under three years of age should therefore be ruled out. Calling a name, or making some familiar sound at close range to the ear will, in a normal child, usually elicit some change in position of the head and toward the source of the sound.

The writer does not regard clapping of the hands, stamping of the feet, a clap of thunder, a locomotive whistle, or similar sounds or noises as adequate tests. It is difficult to determine whether such impressions, if they reach the child, are conveyed by tactile or by hearing sense. Where there is considerable defect in hearing, where nerve deafness is in question rather than a defect in the conducting mechanism, high pitched tuning forks and Galton whistles are of on avail. These sounds would, in the nature of the hearing defect, be minimized or lost because of their high pitch range and their feeble intensity.

The flutter of low pitched tuning forks (32 or 64 d.v.), when held close to the external auditory meatus, might be mistaken for the hearing of a sound; the C forks (128 or 256 d.v.), when testing for bone conduction might also be tactually felt rather than heard.

Much stress has been laid on the use of the palpebral reflex. This consists of a flutter or twitch of the eyelid when a sound of considerable intensity is suddenly produced close to the ear, unsuspected by the subject. If a loud tuning fork, organ tone, sustained voice, or other musical sound be suddenly brought into contact with a given ear, the subject will evince the fact of hearing by a sudden flutter or twitch of the eyelid. This test, however, is not constantly reliable, as congenitally deaf children with some definite evidence of hearing have been found in whom there was no response whatever to the palpebral reflex; another group of deaf children in whom no sound appreciation of any kind has ever been previously observed, showed a positive palpebral reflex reaction. Where there can be so many exceptions to a given rule, such a test must be regarded as of questionable reliability.

The otologist who frequently meets with considerable difficulty in determining whether or not the young child is deaf has several diagnostic approaches in this class that must be given consideration.

The test of the labyrinth, either thermally by douching, or mechanically by rotation, should be applied to determine the degree or absence of nystagmus. Whenever a young child (aged one to four years), profoundly deaf, is found to have a normal nystagmus reaction or even nystagmus of reduced duration, the assumption is justified that the acoustic labyrinth is not entirely without function. Where the static labyrinth shows definitely negative by both caloric and rotation tests and where the functional tests of hearing show no response, the deafness must be regarded as profound and hopeless of acoustic stimulation. This type of patient, then, whether deafness is congenital or acquired in etiology, must be assigned to special training in speech and lip-reading. Modern pedagogy in these fields makes it possible for fluent, understandable speech to be acquired.

The Audiometer. The introduction of the audiometer offers an additional means of determining the presence or absence of residual hearing in congenitally deaf children, the degree and limitations of the defect and the area of the tonal range in which this deafness is most profound. When a congenitally deaf child, tested with the

high powered audiometer intensified to over 100 dcb., no longer evinces tone perception within the area of 300 d.v. to 3,000 d.v., this child, from a utilitarian standpoint, must be pronounced profoundly deaf and with scant hope of reëducation or restimulation by any form of sound amplification, either by voice or instrument.

The ultimate purpose of these special tests in profoundly deaf children is to determine by classification the most satisfactory method of education.

Improvements in the methods of instruction, better understanding of achievement tests, higher qualifications for the teacher of the deaf, the introduction of otologic, psychologic, acoustic and other specialized influences in the study of the deaf child; coöperation on the part of the medical profession legislators, social service agencies and the interested laity—all of these combined influences are tending toward happier, brighter and more efficient opportunities and end results for the congenitally deaf child.

THE HARD-OF-HEARING CHILD

It is quite evident, from the sweeping generalization that there are over 3,000,000 deafened school children in the United States that active steps must be taken to meet this issue. It is even claimed that deafness is on the increase despite the fact that otologic science, genetics, social hygiene and communal welfare are making rapid progress in every direction.

The pedagogic classification of the hard-of-hearing child and his educational development is of even more vital importance than his consideration as a clinical entity. Pedagogically, the classification of the hard-of-hearing child into several groups is dependent on: (a) the age of the child; (b) the degree of defective hearing; and (c) acquired fluency of speech.

There are two types of acquired deafness in children: one, the child has acquired deafness before he has sensed fluent speech; the second type, the child has acquired deafness after fluency of speech has been established.

The child who has acquired total deafness before speech has been developed must be offered practically the same course of training as the congenitally deaf child who has never heard speech. Arbitrarily, this classification is applied to the child who has acquired deafness before the age of three years. Such children may be logically enrolled in a school for the deaf. Where total deafness has been acquired from meningitis, influenza or other infectious diseases in which the toxic selectivity seems to be most virulent in producing profound deafness in children from three to five years of age, intensive, individual training is required. Every effort should be made to conserve speech in fluency, rhythm, pitch, volume and quality. Unless constantly drilled, this type of patient drifts rapidly into slovenliness and inaccuracies of speech and soon acquires the characteristic monotone so prevalent in the congenitally deaf child.

Lip-reading, of course, is an essential factor in the training of such children, for, with the serious loss of hearing, this becomes his most precious asset for social contact. In deafness beyond a range of twelve feet from the source of voice in the schoolroom, the importance of lip-reading and voice conservation by intensive training must be emphasized. A special teacher should be assigned to lip-reading and speech, and the instruction should be continued, preferably in a graded school for hard-of-hearing children. When lip-reading efficiency and fluency in speech warrant, these children may be transferred to the normal schoolroom. The turn-over for the acquisition of lip-reading and speech conservation is accomplished in from one to two years of such intensive training.

In the large cities throughout the country Boards of Education are making provisions for this type of training for hard-of-hearing children, segregating them

into classes for lip-reading and speech conservation, organizing centers where such training may be received and supporting active measures for qualifying specially trained teachers to engage in this field of pedagogy. In the rural communities the problem of giving hard-of-hearing children such opportunities still remains unsolved.

The entire question of the rehabilitation of the hard-of-hearing child is such an important one that ways and means must be found to supply adequate training for this type of physical handicap. The medical profession, especially the otologist, must concern itself more vitally and give of its experience and advice in meeting this issue.

The earliest malpractice suit recorded anywhere was instituted in England in 1767. . . . [It] was an action against a surgeon (Baker) and an apothecary for the alleged mistreatment of the plaintiff's tibia and fibula. From—"The Roentgenologist in Court" by Samuel Wright Donaldson (Thomas).

ON THE DIAGNOSIS AND TREATMENT OF OTOSCLEROSIS*

LOUIS K. GUGGENHEIM, M.D.

LOS ANGELES, CALIFORNIA

LOCALIZED osseous dystrophy of the labyrinthine capsule or histologic otosclerosis, when uncomplicated by tympanal pathology, is not difficult of diagnosis after involvement of the hearing mechanism has occurred. Yet it may exist throughout life and be impossible of recognition if hearing is normal.

Of great importance is the age of onset and the progressiveness of the impairment. Recently a 68 year old man, with hearing markedly impaired, showed a typical nerve deafness audiometric curve with moderate impairment for low frequencies. A so-called senile atrophy of the spiral ganglion would have been the diagnosis had not the family history and the age of onset (22 years) been in favor of otosclerosis. His focus at the site of predilection (anterior to the oval window) was undeveloped, while probably a round window patch had grown into the cochlea in the high tone area. Such cases are not uncommon.

Although otosclerosis is a regressive hereditary malady, we must not lose sight of the fact that otosclerotic bone is under the same influences that control normal bone growth. This explains the clinical manifestation at the end of adolescence. It has been shown by Guggenheim, Bast, Guild, Siebenmann and Manasse that the osseous dystrophy begins many years before an impairment of hearing is noticed. One reason for this is that people are usually not examined until the hearing impairment is greater than the intensity of conversation voice and interferes with the routine of life. In the Deafness Prevention Centre in Los Angeles, where supposedly normal children from 4 to 16 years old are being studied, the very first patient was the "normal" 7 year old daughter of an otosclerotic woman. This patient showed impaired hearing with the audiometer and other signs of an early otosclerosis.

The dystrophic bone constituting the otosclerotic focus is laid down during the fetal period and later is subjected to the same stimuli from thyroid and pituitary hormones that all bone receives. The steady increase in the size of the focus finally results in involvement of the annular ligament, etc.

The pink shimmer sign is seldom seen, but when present is pathognomonic. It is not usually due, as is commonly thought, to the high vascularity of the porous bone of the focus, but rather to the engorged capillaries of the mucoperiosteum of the tympanic cavity overlying the focus.

When uncomplicated, typical otosclerosis manifests the following signs: (1) decrease or absence of cerumen; (2) bilaterality; (3) defective or absent vasomotor response of the drum membrane; (4) normal drum membrane with normal mobility of membrane and malleus; (5) patent Eustachian tubes; (6) negative Rinne test; (7) prolonged Schwabach test; (8) paracusis willisii; (9) varying degrees of tinnitus and throbbing; (10) slight pain in a small percentage of cases; (11) pink shimmer in a few cases; and (12) normal bone conduction with varying degrees of conduction impairment. The air curve may show involvement for lower frequencies and a gradual slant to normal for high tones, but more frequently it is a line straight across. Where the round window and internal meatal foci are present, there may

* From the Department of Otolaryngology, Washington University Medical School, St. Louis and the Department of Physics, University of California, Los Angeles. Research made possible through a grant from the Research Study Club of Los Angeles. Animal experimentation in the laboratories of the Children's Hospital in Los Angeles. Presented before the Research Study Club of Los Angeles, January 1938.

be marked loss of bone conduction. Occasionally air conduction will show a very moderate loss for the lower frequencies and a sudden drop from 2,048 upward. These are atypical cases in which the oval window involvement is insignificant as compared to that of the round window and internal auditory meatus. Such cases are usually diagnosed as nerve deafness.

The family history, when positive, is very important, but the absence of familial deafness should not count against a positive diagnosis when other signs are present, for the following reasons: (1) the patient may not remember; (2) the hereditary disease may have been transmitted by an individual who has himself not manifested impaired hearing; and (3) the father and mother may each have inherited a single character, the patient receiving the two combined, with resulting osseous dystrophy (Davenport—two dominant characters, one sex-linked).

The diagnosis of otosclerosis in which the hearing mechanism has not been involved is obviously impossible. The greatest difficulty arises in those cases of otosclerosis complicated by other pathologies of the ear. Quite frequently the condition is present with various tympanal changes including (1) chronic suppurative otitis media; (2) mild fibrosis due to low grade inflammatory transformations of unresorbed mesenchyme; and (3) fibrosis of purely inflammatory origin.

The recognition of otosclerosis underneath this superimposed pathology may be impossible, but usually bilateral symmetry as to audiometric curve, family history of deafness, age of onset of hearing impairment, and the progressive nature of the affection will permit of a correct diagnosis. When otosclerosis is complicated by an endorgan neural change, the latter may be a part of the otosclerotic picture or may be an independent nerve deafness due to one of many factors. Again the age of onset and family history will often enable one to differentiate the two.

It was once thought that otosclerosis was being diagnosed clinically far too often. Temporal bone sectioning at that time was not so widespread as at present. Today we believe that at least 25 per cent of all cases of deafness are due to this cause. Just what the incidence of silent otosclerosis is, no one knows. Nager found the condition in 5 per cent of individuals succumbing to accidents.

TREATMENT

Of primary importance is the avoidance of propagation by otosclerotics. This should be observed by the otosclerotic woman for two reasons: (1) the offspring may inherit the malady; and (2) the mother may suffer a marked and rapid deterioration of hearing as a result of the pregnancy.

The early recognition of otosclerosis offers the greatest hope for control of deafness from this cause. It is well known that impaired hearing may exist for many years and be progressive without recognition, until the hearing loss is greater than the intensity of the ordinary conversation voice. It was with this thought in mind that the writer established a Deafness Prevention Center.

Until recently otosclerosis has been a scientific curiosity, hopeless from the standpoint of therapy. Certain histologic and physiologic characteristics of the osseous dystrophy intrigued the writer and finally resulted in a therapeutic effort which gives some promise of controlling the progress of the disease. If this is substantiated by further trial and if otosclerosis is diagnosed very much earlier than it has been until now, it would seem that the problem will thus be solved. This contribution has resulted from a study of the histopathology of otosclerosis, its pathogenesis and from a certain clinical fact. The clinical fact: over 50 per cent of otosclerotic women lose markedly in hearing during and after pregnancy.

Otosclerosis is a progressive form of deafness because the foci of abnormal bone anterior to the oval window, at the

round window and at the internal meatus, have the tendency to grow larger. The increase in dystrophic bone is at the expense of the original capsular bone, except for the hyperostotic bulges into the tympanum and labyrinthine spaces. Around the dystrophic bone, capsular bone degenerates, losing its calcium and phosphorus, and finally becoming completely resorbed.* If this did not occur, it is rather certain that the focus of otosclerosis would not grow larger and that deterioration of hearing could not develop.

Clinically and experimentally it has been shown that areas of bone resorption can be reconstructed. Alveolar process resorption may be reversed by the administration of calcium-phosphorus and the necessary vitamins. Areas of bone resorption in osteodystrophia fibrosa, resulting from excessive parathormone, will recalcify and appear normal in the *x*-ray after the adenoma of the parathyroid has been removed.

The resorption of bone in otosclerosis is probably due to pressure, interference with circulation, the presence of ferments, oxidation phenomena, osteoclasts, blue mantles, etc.

Otosclerosis is classed among the incurable diseases. Described as a pathologic entity many years ago, it has been something to diagnose and nothing more. True, otologists have come to realize that there is a psychic factor and that their responsibility has not ended until this has been dealt with and until pregnancy has been advised against, lip-reading urged, and, when necessary, a proper hearing aid selected. Although various therapeutic measures have been proposed there is almost universal agreement that none of these has any virtue.

The removal of the foot-plate was tried in Politzer's day, but the operation was abandoned because the oval window promptly closed with bone. Barany and others created artificial fistulae of the canals and reported marked improvement in hearing of several weeks' duration. Invariably the fistulae closed and the impairment reverted to the former level or hearing was completely lost. In 1933 Gruner Holmgren revived the fistula operation with a new technique. In 1935 he reported a number of cases with marked improvement in hearing which had persisted for two years. In 1937, when Holmgren was asked for final information as to these cases, he replied (in a personal communication) that all had again lost their hearing and that his experimental animals, without exception, showed that the fistulae promptly closed with new bone.

Non-surgical treatment has included innumerable drugs and procedures all without influence upon the disease.

One report is particularly interesting because it is just the opposite of the experimental study under discussion. Crockett and Aub's decalcification treatment had for its object the removal of calcium from the oval window region in early otosclerosis. It was reasoned that if calcium could be withdrawn from the annular ligament and foot-plate before firm ankylosis had occurred, the continued conduction of sound through the oval window could be maintained. A number of otosclerotics were placed on a calcium-free diet and given parathormone and ammonium chloride. Fortunately for the patients, this treatment was abandoned before harm had resulted to the general osseous system. The fallacy of the experiment was two-fold. The decalcifying régime could not possibly act upon a minute area in the aural capsule, the densest bone in the body, without also producing a generalized osseous pathology such as we find in osteodystrophia fibrosa. Secondly, even if calcium had actually been withdrawn solely from the otosclerotic focus, the collagenous mass remaining would promptly have taken up that mineral again to reform the focus the moment treatment was discontinued.

* In a calcium research with white rats now under way, it has been noted that the osteocytes in resorbing bone are mobilized as fibroblasts.

An understanding of the present thesis requires some knowledge of the normal structure of the capsule and its ontogenesis, the biochemical phenomena of bone formation and resorption and the pathogenesis of otosclerosis.

STRUCTURE OF THE OTIC CAPSULE

In the adult capsule are three types of bone: (1) fine-fibered, lamellarless or skein bone; (2) lamellar bone; and (3) calcified lacunae or so-called cartilage rests. The endosteal layer, immediately surrounding the membranous labyrinth, is a membrane bone of lamellarless structure. The outer layer of the capsule is a perichondrially derived membrane bone of lamellar and lamellarless structure. Between the periosteal and endosteal layers is the enchondral or cartilage bone, showing calcified lacunae, skein bone and some lamellar bone. These three layers are of regularly formed, orderly bone. It is in the middle layer that otosclerosis starts.

ONTOGENESIS OF THE OTIC CAPSULE

In an embryo of 28 days the auditory vesicle is surrounded by condensed mesenchyme—the first evidence of capsule. By the sixth week the mesenchymal elements have differentiated into large chondroblasts with dark staining nuclei. These cells are rich in chromatin granules and are connected with each other by heavy protoplasmic processes. No intercellular substance is present. By the eighth week it is noted that many of these processes have been resorbed, the remainder coalescing in such a manner as to form immature cartilage lacunae. The chondroblasts have become cartilage cells. A very light basophilic matrix appears, but as yet there is no perichondrium. By the third month perichondrium has formed and matrix has matured and we find the aural capsule of dark staining adult cartilage. Just before the fourth month, ruptures occur in the perichondrium and through them pass into the capsule, osteogenic capillary buds accompanied by osteoblasts and histiocytes. These elements accomplish an excavation of the cartilage, leaving finally no vestige of cartilage except the calcified lacunae. These act as anchor islands for the support of osteoblasts and are finally surrounded by fetal enchondral bone. At the same time, from the inner layer of perichondrium, certain cells form the first layers of periosteal bone, while around the membranous labyrinth there forms from membrane, the endosteal layer. All the fetal bone is in apposition to hematopoietic marrow. Both the fetal bone elements and the blood-forming marrow are destined to disappear. They are replaced by the types of adult bone before mentioned and a fibro-fatty marrow.

Throughout life constant resorption of capsular bone with the formation of new bone is occurring in all but the enchondral layer where the vital processes are almost entirely absent after fetal bone has been replaced by that of adult form. The most active alterations, normally, and in most pathologic conditions, occur in the periosteal layer. When old age arrives, resorption in the periosteal layer continues but replacement fails, so that we find, then, very large marrow spaces filled mainly with fat.

BIOCHEMISTRY OF BONE FORMATION AND RESORPTION

Going back to the condensed mesenchyme of the 4 weeks embryo, let us discuss the biochemistry underlying its morphologic differentiations. Little is known of the *vis a tergo* of mesenchymal differentiation into chondroblasts at the sixth week. We know only that the genes for capsular development act in the presence of maternal nourishment and adequate oxidation. Even a very temporary interference with oxidation will result in malformation. Also hereditary factors such as exist in otosclerosis, osteogenesis imperfecta, chondrodystrophy, etc., will determine abnormalities of differentiation. Further, certain deficiencies, notably of

vitamin C, will interfere with normal development.

Just before the eighth embryonic week and in the presence of adequate vitamin C, the chondroblasts secrete an intercellular substance, distinctly fibrillar under high magnification but seemingly homogeneous under low magnification. This intercellular substance, which is basophilic, is deposited around, not within, the young cartilage lacunae. As the cartilage matures, and with ordinary stains, the fibrils disappear. With silver stains and polarized light, however, fibrils may still be found.

In the mature cartilage cells at three months are found fat and glycogen. Thread-like mitochondria, sometimes in the form of granules or short rods, are noted.[1] To one side of the nucleus is the Golgi apparatus of coarse network. Usually there are two nucleoli. Matrix is composed of collagen, chondromucoid, chondroalbumoid and chondroitin-sulfuric acid. The basophilic staining of matrix is due to the presence of the chondroitin sulfuric acid. The chondromucoid also participates. The collagen is itself acidophilic, but its reaction is masked by the two substances mentioned. Growth of cartilage is by mitotic division in the chondrogenic part of the perichondrium and by mitotic or amitotic division within the lacunae. There are no blood vessels in cartilage; nourishment seeps through from the perichondrium. There are no nerves and no lymphatics, although interlacunar canaliculi have been described.

When, just before the fourth fetal month, perichondrial ruptures occur and osteogenic buds of mesenchymal origin pass in, the cartilage cells enlarge and distend the lacunae. At this time the cartilage cells and osteoblasts produce the enzyme phosphatase which is concerned with ossification. Next the cartilage cells become pyknotic and disappear. In the matrix are deposited calcium salts and magnesium from body fluids. This causes the matrix to take the hematoxylin stain more darkly and gives a gritty feel to the cartilage upon section. Rapid destruction now ensues. Within certain persisting cartilage lacunae, the cartilage cells are replaced by osteoblasts. Calcium is deposited and there results an ossified lacuna which, giving the impression of cartilage, has come to be called a cartilage-rest. Myriads of osteoblasts swarm into the spaces left by cartilage destruction. They arrange themselves in epithelioid alignment along the edges of the persisting lacunae and begin to secrete collagen. The osteoblasts become the osteocytes, and another row of osteoblasts forms. This process continues until, with the addition of calcium, phosphorus and magnesium, red-staining fetal bone is evolved in the form of the original cartilage model. Marrow spaces, large in the beginning, are filled with blood forming elements.

Around the membranous labyrinth, mesenchymal elements have formed a narrow band of membrane in which osteoblasts produce endosteal bone. On the inner surface of the perichondrium, cells wander forth as perichondrial osteoblasts to lay down the membrane bone of the periphery of the capsule. As bone formation increases, the large hematopoietic marrow spaces grow smaller. The vessels of the osteogenic buds become the vessels of the Haversian system.

Bone matrix consists of organic and inorganic constituents. The organic is bone collagen or ossein, mucoid and an albuminoid (osseomucoid and osseoalbumoid.) Sixty to 70 per cent of the total is inorganic, consisting of calcium and phosphorus with a small amount of magnesium. Bone matrix is fibrillar. The fibrils are variously arranged in bundles.

From the inner layer of perichondrium and from mesenchyme, are formed, in addition to osteoblasts, multinuclear cells called osteoclasts which are concerned with destruction of bone and with phagocytosis of osseous débris. The osteoblast is basophilic, the osteoclast, oxyphilic. With phosphotungstic acid the osteoclast is granular. Its cytoplasmic border is usually

irregular, often showing comb-like processes protruding into the bone which is being resorbed. These indentations are called Howship's lacunae. The osteoclast, thought by some to be purely phagocytic, does not take the vital stains.

Throughout life, bone is being formed by osteoblasts and destroyed by osteoclasts and other factors. It is thought that carbon dioxide produced by living cells participates in the dissolving of dead bone. Bone is a storehouse of calcium which is readily released when the need arises in any part of the body. Marked loss of calcium may be followed by complete resorption of bone. Bone may also be destroyed by creeping replacement through osteoblast invasion. There is probably some chemical effect from osteoblastic secretion,* from tissue fluids as well as from direct action of osteoclasts.

Haversian canals contain blood vessels, lymphatics, osteoblasts and other cells. The fibrils of the Haversian lamellae run in different directions to give architectural strength to this form of bone. Haversian bone is true lamellar bone, one layer of bone, one layer of cementum and so on. From the bone cells, small canals pass into the matrix. They run in all directions, anastomosing with canaliculi of other cells. Through these canals nutrient fluids pass. Periosteum is anchored to bone by collagenous bundles, Sharpey's fibers. Nutrient paths from the periosteum are called Volkmann's canals. Periosteal lymphatics accompany the vessels in these canals. Blood vessels are numerous in the labyrinthine capsule.

CALCIUM AND PHOSPHORUS

The essence of the problem of otosclerosis is bone. The focus of otosclerosis is bone. The resorbing capsule around the focus is bone. If we are ever to understand the problem and if we are ever to find a therapeutic agent of worth, we must start with an understanding of bone. Generally the attitude toward the supporting frame of the body is that it is a structure which is fixed and permanent unless broken. Let us then begin by understanding that bone is constantly changing—constantly being destroyed and reformed, in health as well as in disease. Its function is not only to support the body but to act as the great storehouse of calcium and phosphorus, ready to supply, in emergency, these elements to whatever part of the body is in need. Rapid mobilization of calcium is accomplished through the action of parathormone. Although we are concerned mainly with the deposition of calcium and phosphorus in the aural capsule, we should know something of the other functions of these minerals, since we are to administer rather large amounts of them to our patients.

In addition to its rôle in the development and maintenance of tooth and bone structure, calcium is concerned with coagulation of blood and milk, in neuromuscular irritability, membrane permeability, heart action, enzyme activation, allergy and in the utilization of iron.[2]

Phosphorus, in addition to its importance as a constituent of bone and teeth, helps to maintain the acid-base equilibrium of the body, is an important component of all nerve tissue, participates in the mechanism of muscular contraction and is concerned with carbohydrate metabolism. Calcium and phosphorus are indispensable in the reversal of halisteresis, but they cannot possibly accomplish this desired change in the otic capsule until they are absorbed and metamorphosed into physiologically utilizable form. It is, then, not sufficient merely to prescribe calcium and phosphorus; we must be assured that they will be absorbed and deposited in the decalcified areas of the temporal bone. After giving consideration to the normal requirements as supplied in such foods as cheese, milk, etc., the necessary additional amount is best given in the form of dical-

* Decalcification, particularly with ammonium chloride has been characterized by a halo of matrix loss immediately around the osteocytes in advance of the calcium loss elsewhere.

cium phosphate. This should be taken a half hour before breakfast or just before retiring; never at meal time, as absorption of calcium requires an acid medium.

The absorption of calcium takes place mainly in the upper portion of the small intestine in an acid medium produced by the not yet neutralized hydrochloric acid from the stomach. Farther down, where the contents are alkaline, it is not absorbed. Lactose, by producing lactic acid in the intestine, will aid in the process. This substance is contained in milk, but additional amounts of pure lactose or whey are also prescribed. Since calcium phosphate and calcium carbonate are practically insoluble in alkaline solution, they will, when placed in such a medium, be precipitated as insoluble calcium soap and be excreted. When the acid gastric juice enters the small intestine, calcium must be absorbed before neutralization takes place.

Diets containing excessive phosphorus in proportion to calcium interfere with calcium absorption because of the formation of nonabsorbable tricalcium phosphate. Likewise, too much fat in the intestine will result in the formation of insoluble soaps of calcium and fatty acids, which are excreted without any absorption. Such a situation may result from excessive amounts of fat in the diet, from inadequate bile or from a metabolic disturbance, as in celiac disease. Essential to calcium absorption and utilization is vitamin D. Absence of bile will interfere with the absorption of vitamin D and thus in another manner interfere with calcium utilization.[3] Large amounts of certain oxalate-containing vegetables, such as spinach, may interfere with calcium absorption through precipitation.

Phosphorus is absorbed as phosphates from the intestine and through the same influences as in the case of calcium. Excessive amounts of calcium will interfere with phosphorus absorption through the formation of insoluble tertiary phosphate.

When finally calcium gets into the blood it is found in the serum as diffusible calcium and in the non-diffusible form, bound to protein. It is only the diffusible form (mainly positive calcium ions) which participates in the various physiologic functions of the element; the protein-bound constitutes an ever ready source upon which the body may draw. This non-diffusible calcium proteinate results from the calcium positive ion acting as a base in relation to protein. A colloidal form of calcium phosphate is formed when the blood stream is suddenly flooded with positive calcium ions, as for example, in intravenous injection of calcium or when bone calcium is rapidly mobilized, etc.

From the blood, calcium and phosphorus are deposited in bone and teeth mainly as calcium phosphate and carbonate. There is normally a constant equilibrium between the transport calcium-phosphorus in the blood and their solid phase in bone. The total serum calcium is 9 to 11.5 mg. per 100 c.c., with an average of 10. The diffusible fraction varies from 4.5 to 5.5 mg. per 100 c.c., and the non-diffusible from 4.1 to 7.2 mg. An increase of phosphorus may mean depressed calcium, especially at the expense of the positive ions. A non-diffusible calcium phosphate forms and is removed from the blood stream. For this reason the ratio of calcium to phosphorus is of great importance in efforts at recalcification.

FACTORS INFLUENCING CALCIFICATION

All those elements of which bone is composed must be ingested in adequate amounts and under conditions which will facilitate absorption from the alimentary canal.

Magnesium is adequate in all diets, and usually phosphorus is also, but many diets are totally inadequate in calcium. Protein to form bone matrix (collagen or ossein) is usually adequate. Vitamin C is indispensable to the jelling of collagen, while the absorption of calcium from the intestines and its deposit in bone requires an adequate amount of vitamin D. Vitamin A is important in maintaining the integrity of intes-

tinal epithelium, and vitamins B and G in maintaining intestinal tone. A normal functioning of the parathyroids assures a proper concentration of serum calcium; in addition, thyroid secretion is essential to calcification.

If these various elements are present in optimal amounts, then the larger the calcium intake (within certain limits) the greater will be the retention and utilization in the formation of bone. If amounts of calcium and phosphorus, very much larger than the requirements are given to a well individual there occurs simply an excretion of the excess. As before stated a proper ratio between calcium and phosphorus is important. The normal food ratio is approximately one of calcium to one and one half or two of phosphorus. Diets containing excessive amounts of phosphorus in proportion to calcium diminish the absorption of calcium by the production of nonabsorbable tricalcium phosphate. Where resorption of bone is occurring anywhere in the body, increased retention of calcium will tend to heal (recalcify) the areas of resorption.

Toxic doses of vitamin D may cause hypercalcemia (serum calcium 15 to 20 mg. per 100 c.c.) and hyperphosphatemia (serum phosphorus 10 to 12 mg. per 100 c.c.). When serum calcium is maintained above 15 mg. per 100 c.c. metastatic calcification may occur.

Toxic amounts of parathormone, as in adenoma of the parathyroids, will cause hypercalcemia (serum calcium of 12.1 to 23.6 mg. per 100 c.c.) and increased urinary excretion of calcium. Normally 10 to 30 per cent of the calcium is excreted in urine, and 70 to 90 per cent in the feces. In hyperparathyroidism this may be reversed. On the other hand, serum phosphate is subnormal (1 to 3.5 mg. per 100 c.c.), and there is an increased urinary excretion of phosphorus. Calcium and phosphorus are thus withdrawn from bone, the excessive parathormone raising blood calcium by taking it from bone. Excessive amounts of vitamin D, on the other hand, raise serum calcium by increased absorption from the intestine.

Dr. Lawrence E. Detrick of the University of California, in a personal communication, stated that from his recent experiments with vitamin C it seems possible to accomplish the physiologic utilization of calcium with adequate amounts of that vitamin even in the absence of vitamin D.

The utilization of radioactive calcium and phosphorus in the treatment of otosclerosis might offer a method of visualizing the progress of recalcification were it not for the fact that the peculiar characteristic lasts too short a time, in the case of calcium, only about two hours.[4]

PHOSPHATASE

This enzyme, secreted by osteoblasts and cartilage cells, invites the deposit of calcium in collagen by breaking down phosphoric esters, thus increasing the concentration of phosphate ion, which with calcium forms insoluble calcium phosphate to be deposited in the ossifying centers. Phosphatase is present in adult serum to the extent of 1.5 to 4 units per 100 c.c. In children, where more active bone changes are occurring, the average is 7 units. The amount is greatly increased in certain diseases, notably rickets, osteodystrophia fibrosa von Recklinghausen, etc. For the normal functioning of phosphatase, magnesium is required. About 1 per cent of bone ash consists of the latter.

RELATIONSHIP OF CALCIUM, PHOSPHORUS, SODIUM AND POTASSIUM

Calcium decreases neuromuscular irritability; sodium and potassium act as stimulants. Excessive amounts of sodium and potassium are detrimental to normal calcification and result in increased urinary excretion of calcium. Excessive amounts of phosphorus result in increased excretion of calcium, as does also ingestion of mineral acids.

Seventy-five per cent of the total ash of the body consists of calcium phosphate.

Of the total body calcium, 99 per cent is in bone, 1 per cent in soft tissue and fluids. The total body phosphorus is apportioned as follows: in bones and teeth 70 per cent; in muscle approximately 29 per cent; in the brain and nerves, approximately 1 per cent.

Not much is known of magnesium metabolism. Of the body total, 71 per cent is found in bone, the remainder in the soft tissues. Muscle contains more magnesium than calcium, while in blood the opposite holds. Sodium and potassium are present mainly as chlorides. They function as soluble salts in the body fluids, as the producers of alkalinity or acidity of the digestive juices, etc., and to maintain osmotic pressure.[2] There is an antagonism between sodium-potassium and calcium-magnesium. A high intake of sodium tends to increase potassium elimination, but calcium aids in correcting a disturbed equilibrium between sodium and potassium. In neuromuscular irritability the equilibrium between calcium on the one hand, and sodium-potassium on the other is most important.

DIET ANALYSIS

Practically all human diets are markedly deficient in vitamin D, so essential to the utilization of calcium. Therefore, when we undertake to preserve the integrity of bone and teeth and to reverse abnormal resorption of bone, as in otosclerosis, there must be assured more than the usually adequate amount of this vitamin. Although vitamin D is produced in the body by the action of sunlight upon skin ergosterol, we have no means of determining the amount so produced. The richest supply of vitamin D is fish oil and irradiated ergosterol (viosterol).

Vitamin D, adequate calcium and phosphorus are essential in the preservation of osseous integrity, but protein and vitamin C are also indispensable, for the matrix of bone cannot form at all without them.

How are we to determine whether or not a patient is getting these various factors in adequate amounts? The inaccurate way is merely to ask the patient what he eats, then attempt to figure out whether his diet is correct. The scientific way is to hand the patient a printed form with printed directions. The form should have a line for each item of food taken and columns for the amount taken for at least seven days. All food, drink and medicines, at meals and between meals, must be included. When the form is completed, the next step is to break each item down into its elements. For each separate item of food, the content of protein, carbohydrate, fat, calories represented, the calcium, phosphorus, iron, iodine, copper, acid-base ratio, vitamins A, B, C, D, G, etc., is calculated, and the average daily intake computed.

By comparison with the daily requirement, one can see what corrections are necessary. This correction of diet would be simple were it not for the fact that the overweight and underweight patients present certain further needs, allergic states require consideration, climatic conditions alter requirements, etc.

Diet analysis is time consuming and the busy practitioner had best turn it over to a dietician. It is, however, an intriguing and instructive undertaking and results in a far better grasp of the significance of food factors than one gets from the perusal of a dietician's conclusions. The process of diet analysis and determination of individual requirements has been greatly simplified by an ingenious invention of Michael Walsh of San Diego.[5] This consists of two revolving discs with practically all foods noted at the periphery. One turns an arrow to the particular food and has only to read off on the di-calator, the amounts of protein, carbohydrate, etc., represented. Even with this device, an analysis of a seven day diet requires many hours and much thought.

PATHOGENESIS OF OTOSCLEROSIS

Almost all specimens of otosclerosis from adults show bilaterally symmetric, con-

fluent foci of porous, reticular bone at the site of predilection. (Fig. 1.) Some show, in addition, a similar focus at the round window and internal meatus. The blue-staining focus shows a sharp line of demarcation from the surrounding capsular bone; its fibers end abruptly and never merge with those of the capsule. The porosities are marrow spaces most of which are quite characteristic. At the center is an engorged capillary surrounded by numerous large round and oval osteoblasts. Rarely are these osteoblasts found in epithelioid alignment as in normal bone formation. In addition, there are histiocytes, undifferentiated mesenchymal elements and fibroblasts. As new reticular bone is formed at the periphery of the marrow spaces, these spaces grow smaller. A few osteoclasts are found in some specimens while in others they are entirely absent. Growing from the focus into the surrounding capsule are blood vessels surrounded by blue staining cementum. These are the blue mantles.

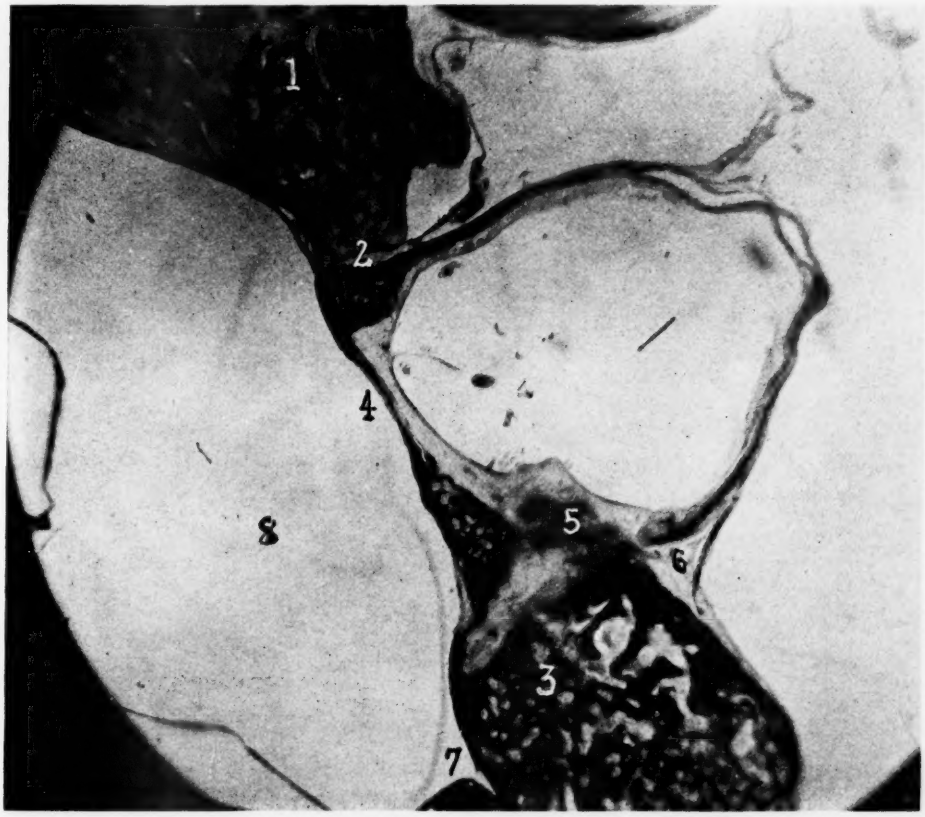


Fig. 1. Typical otosclerotic focus at site of predilection, involving annular ligament, foot-plate and anterior crus. (1) Normal capsule. (2) Annular ligament, posteriorly. (3) Otosclerotic bone. (4) Foot-plate of stapes. (5) Projection of dystrophic bone through anterior crus. (6) Characteristic band of connective tissue. (7) Fissula ante fenestram. (8) Cisterna perilymphatica.

The focus may be very small, not involving the hearing mechanism, or it may have produced ankylosis of the stapes, closure of the round window, and extensive involvement at the internal meatus and pericochlear region, with varying degrees of hearing loss.

Surrounding the focus of otosclerosis is a condition of the capsule which is the basis for the therapeutic experiment presented. This condition is one of halisteresis. Preparedness or halisteresis around the otosclerotic focus (Fig. 2) has been recognized for many years, but its utilization in the treatment of otosclerosis is original with

the writer. Calcium has been withdrawn (Fig. 3), and the osteocytes are being mobilized as fibroblasts. Decalcification Pressure exerted by the expanding focus, interference with capsular circulation, osteoclasts, etc., are probable factors.

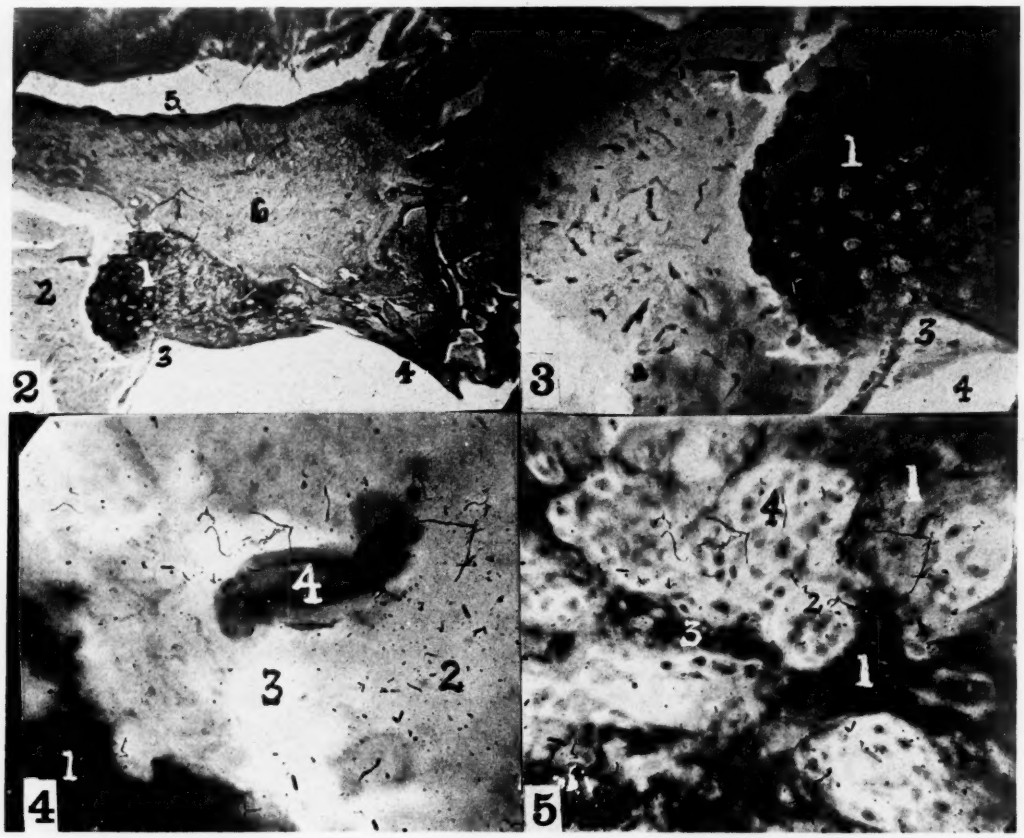


Fig. 2. Otosclerosis complicated by otitis media purulenta. (1) Otosclerotic bone. (2) Aural capsule undergoing decalcification. (3) Fissula ante fenestram. (4) Foot-plate of stapes. (5) Tympanal epithelium. (6) Fibrosis of tunica propria (otitis media purulenta).

Fig. 3. Focus in Figure 2 under higher magnification. (1) Otosclerotic bone. (2) Decalcifying capsule. (3) Fissula ante fenestram. (4) Cisterna.

Fig. 4. Blue mantle from focus of otosclerosis. (1) Otosclerotic bone. (2) Decalcifying aural capsule. (3) Resorption area. (4) Blue mantle.

Fig. 5. Giant cell osteoclast in a focus of otosclerosis. (1) Otosclerotic bone. (2) Osteoclast. (3) Otosclerotic bone. (4) Marrow space.

continues until a resorption area (Fig. 4) invites the invasion of osteogenic marrow from the otosclerotic focus. (Fig. 5.) Dystrophic bone formation follows and thus extension of the focus with further impairment of hearing occurs. What causes the normal capsule to degenerate and be resorbed? It has long been thought that the blue mantles participate, either by pressure or biochemically. An interesting theory, recently suggested by Dr. Lewis Gunther, is that the blue mantled vessels withdraw calcium from the normal capsule and deliver it to the growing focus.

What is the initial pathogenetic phase of otosclerosis? In a seven months fetus[6] in the region of the round window, scattered blue-staining foci of dystrophic bone were found. The usual hematopoietic marrow had here been converted into a fibrous type. (Fig. 6.) At the edges of the marrow spaces, reticular, blue-staining bone was being laid down. Capsule in the vicinity showed marked halisteresis and was being invaded by the weed-like osteogenic marrow. Cartilage rests were undergoing destruction. If this specimen is proved to be the first stage of otosclerosis, then the

process begins in marrow and later displaces capsule.

In a case reported by Guild,[7] the focus continues to undergo degeneration and resorption, permitting the focus to increase in size. One cannot conceive of any therapy

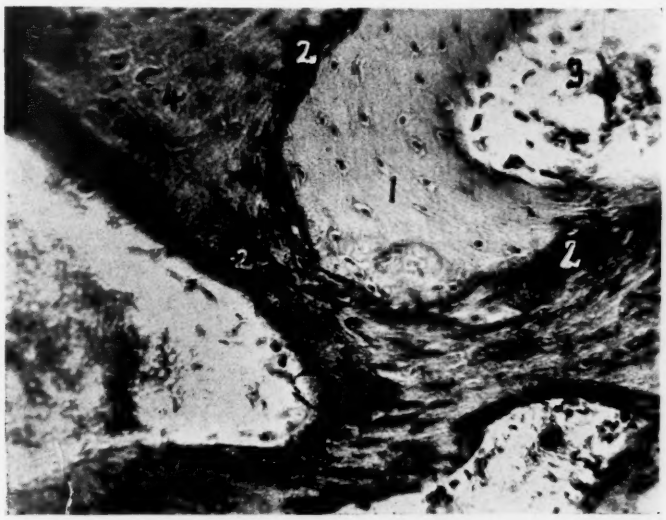


Fig. 6. Otosclerotic focus in a seven months fetus. (1) Normal fetal bone with area of halisteresis at lower edge. (2) Blue-staining dystrophic bone. (3) Fibrous marrow. (4) Halisteresis of fetal bone.

near the oval window, instead of being a confluent mass, was composed of a number of small, scattered foci. There was also a large cartilage rest anterior to the oval window. Many spaces were filled with fibrous tissue in which were present plump cells. Adjacent to the normal bone were a number of osteoclasts. This patient, one year and ten days old, would seem to present a transition between the seven months fetus and the usual adult case of otosclerosis. Bast[8] has also reported foci of dystrophic bone in the ears of several fetuses and believes these foci may represent the earliest phase of otosclerosis.

Summarizing, it would seem that otosclerosis begins as a fibrous marrow displacement of the hematopoietic type, normal in the fetus. As this fibrous marrow forms reticular bone, the surrounding capsule is in some manner adversely affected and finally undergoes halisteresis. Into the resorption areas extends the osteogenic marrow. As several small areas grow larger they finally coalesce to form a confluent focus. Even this confluent focus is not at rest. The surrounding capsule which would destroy the dystrophic focus and bring about a filling in with normal bone, but there is clinical experience and experimental evidence suggesting the possibility of reversing the halisteresis surrounding the focus, thus inhibiting the expansion of the dystrophic area.

It has been proved that an adequate supply of calcium and sufficient vitamin D to ensure its utilization, will help to prevent dental caries and resorption of the alveolar process. Further, it has been shown that the alveolar bone can be recalcified if resorption is not too extensive. Analogous to alveolar recalcification is a treatment of rachitic deformity now being used. In rickets there is imperfect calcification of bone due to calcium and vitamin D deficiency, etc. The incompletely calcified bone or osteoid is soft, thus producing the characteristic deformities. Such cases are now further decalcified, the deformed and softened bones straightened, then reconstructed with calcium, phosphorus, vitamin D, etc.

Otosclerosis begins in embryonic life, the osseous dystrophy appearing shortly after

the onset of ossification of the labyrinthine capsule, but the clinical recognition of the malady does not usually occur until adulthood and not until there has been a rather marked loss of hearing. One reason for this is that no systematic attempt has been made to test accurately the hearing of seemingly normal children, individually and in a sound proof room. Particularly should the children of otosclerotics be so tested.

In addition to the progressive characteristic of otosclerosis, two outstanding clinical phenomena have been recognized for many years: (1) Deafness usually first manifests itself distinctly enough to be recognized by the patient and his family, in late adolescence, the end of the period of greatest growth (bone change) and the period of greatest endocrine activity. The focus of otosclerosis, being bone, is subject to the same stimuli as normal bone, namely pituitary and thyroid influence. The extension of the focus from a silent area into the annular ligament and footplate brings the condition to recognition. The enormous demand for calcium and vitamins during adolescence is often not adequately supplied, so that some withdrawal of calcium from the capsule doubtless occurs. Resorption around the focus plus endocrine stimulation of the reticular bone results in the spread of the dystrophic area.

(2) Over 50 per cent of otosclerotics show a marked loss of hearing following childbirth and some during the latter half of pregnancy. This phenomenon has never, to the writer's knowledge, been satisfactorily explained. In attempting to evaluate it, one must visualize the focus of otosclerosis and understand certain phases of the maternal metabolism.

We are now familiar with the focus of dystrophic bone surrounded by degenerating capsule. That a stimulus to dystrophic bone activity results from the increased endocrine activity during pregnancy seems probable. Seemingly, however, the outstanding factor is the enormous withdrawal of calcium from the mother to supply the osseous elements of the fetus and later the calcium in her milk. It is the writer's belief, established after questioning a number of obstetricians, that the amount of calcium administered to pregnant women, particularly otosclerotics, is inadequate and that the calcium is not given early enough.

The favorite form for calcium administration is calcium gluconate, which is not ideal, because the gluconate molecule is very large as compared to that of calcium. The calcium dose is therefore inadequate. Although less soluble, dicalcium phosphate supplies much more adequately the calcium and phosphorus needs of the mother and should be given in large doses. When the pregnant and later the nursing mother is not receiving an adequate amount of calcium, she uses her own bones to supply the need. The degeneration of capsule around the otosclerotic focus is increased and into the enlarging areas of resorption, osteogenic marrow grows. In this manner the focus rapidly enlarges and marked deterioration of hearing may follow.

THE THERAPEUTIC EXPERIMENT

It is a fact easily demonstrable that around the focus of dystrophic bone in otosclerosis the capsule is in a state of so-called preparedness. That is, capsular bone is losing calcium. It is known that the enlargement of the focus depends upon this halisteresis of the capsule. Hypothetically, an inhibition to the enlargement of the focus would result from a recalcification of the perifocal preparedness area.

Obviously little could be gained by instituting treatment in any case with already marked impairment of hearing; the ideal case for treatment would be one with minimal impairment. Since there is no thought of improving existing hearing, cases for treatment should be selected with the utmost care, and audiometry should be repeated every two months. Supervision of medicines and diet must be constant; it is human for enthusiasm to

lag and carelessness to develop whenever any very prolonged dietary control and medication is attempted.

Proof of virtue in this treatment of otosclerosis must needs depend upon a large series of patients and upon animal experimentation. To state after the writer's one year of experimentation that the treatment has succeeded because a few otosclerotics have shown no deterioration in hearing would obviously be unscientific—otosclerosis is a temperamental malady which may run for many years without loss of hearing and then within a few months show a marked change for the worse. Necessarily, several hundreds of these cases will have to be under treatment for a number of years before the evidence of success can be accepted.

Treatment to produce a reversal of halisteresis consists of:

1. Analysis of diet.
2. Correction of diet to normal requirements for age, type of work, etc.
3. Vitamin C, 1,000 to 3,000 international units daily, in the form of orange juice, lemon juice and tomato juice; occasionally cevitamic acid.
4. Calcium, 3 to 6 Gm., and phosphorus, 6 to 12 Gm. daily in the form of dicalcium phosphate.
5. Vitamin D, 3,000 to 6,000 U.S.P. units daily, mainly in the form of fish oil fortified with natural vitamin D.
6. Where tinnitus is present, 1,000 to 2,000 Sherman units of vitamin B_1 in the form of brewer's yeast, vitamin B complex, or crystalline vitamin B_1 (synthetic).

Experimentally we must prove that the mammalian capsule can be decalcified and later recalcified. With this in mind a staff was appointed by the Research Study Club of Los Angeles to carry out a calcium research upon white rats. This staff, consisting of Dr. Lewis Gunther, Mrs. Mary Irvine, Dr. Victor Goodhill and the writer, has just completed the first half of its experiment, which consisted of various methods of producing halisteresis of the otic capsule. The second half will attempt to produce decalcification of the otic capsule and then reverse it. In addition to the histologic study of the ears, the remaining bones of the body will be biochemically analyzed.

REFERENCES

1. Ham, Arthur W. In Cowdry's Special Cytology, vol. II. New York, Hoeber.
2. Walsh, M. J. Applied Nutrition. Lecture III before the Los Angeles Dental Association, 1937.
3. Gunther, Lewis. Address before the Endocrine Study Group of Los Angeles, Jan. 13, 1937.
4. Greenberg, David. Personal communication from the Dept. of Chemistry, University of California.
5. Walsh, Michael J. Diet analysis. *J. Am. Col. Proct.*, 8: 10 (June) 1936.
6. Guggenheim, L. K. Otosclerosis in a seven months fetus. *Ann. Otol.*, 42: 690–691 (Sept.) 1933.
7. Guild, S. R. Early stages of otosclerosis. *Arch. Otolaryng.*, 12: 457–483 (Oct.) 1930.
8. Bast, T. H. Development of the otic capsule etc. *Arch. Otolaryng.*, vol. 23 (May) 1936.
9. Guggenheim, L. K. Monograph on Otosclerosis. St. Louis, 1935. Published by the author.

MALIGNANT DISEASES OF THE PARANASAL SINUSES

GORDON B. NEW, M.D.

Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic

ROCHESTER, MINNESOTA

IT is safe to say that in the last fifteen years there has been no greater advance in treatment of malignant tumors than in that of malignant growths of the upper jaw and sinuses. The purely surgical methods, dissection by knife and scissors, employed twenty years ago in resection of the upper jaw, have been replaced by the various types of cauterization, surgical diathermy and the use of radium and Roentgen rays.[1] In the treatment of these tumors the objective must be complete eradication of the tumor at one time, regardless of the resultant deformity. Loss of an eye or perforation of the cheek must be considered of secondary importance. Plastic surgery can correct these to a great extent if the tumors do not recur.

DIAGNOSIS

Physicians in general should know that a dull, persistent pain in the face or upper jaw, usually made worse by lying down, may be attributable to a malignant tumor of the sinuses. Recent nasal obstruction or obstruction of the lacrimal duct, increased discharge from the nose or purulent discharge with nasal bleeding should cause the possibility of malignant tumor to be kept in mind until the condition has been proved to be benign. If, after application of the usual methods of examination, including roentgenography, there is still doubt as to the possibility that a malignant lesion is present, exploration of the antrum or sinuses should be carried out so that a fresh, frozen section of the tumor may be examined. Too often conservative treatment is employed and the diagnosis is not made until bulging of the cheek and eye renders the condition self-evident.

The situation of the tumor determines the early symptoms. Dentists should know that persistent loosening of some of the teeth may be the first symptom of a malignant tumor. Frequently teeth are removed for what is thought to be pyorrhea, but later it is found that an antral tumor, bulging down from above, has loosened the teeth. Nasal obstruction, increased nasal secretion and obstruction of the lacrimal duct may be caused by a tumor of the lateral wall of the nose and ethmoid region. Bulging of the cheek of short duration, with pain, affecting a person of middle age, is practically always owing to a malignant tumor if the usual inflammatory conditions associated with the teeth have been excluded. Inflammatory conditions of the antrum itself do not produce bulging of the cheek unless osteomyelitis is present. The swellings and infections about the teeth usually are readily diagnosed.

Frequently patients are referred for treatment after exploration of the antrum has been made and after a pathologist, on the basis of biopsy, has reported that the condition is malignant. In such cases it usually is difficult for the surgeon to whom the patient is referred to determine the extent of the original condition, how much of the swelling is the result of the exploration and how much is attributable to the malignancy. In general, the patient's chance of getting well is much better if, at the primary operation, a fresh frozen section is examined and, provided the condition is found to be malignant, the necessary treatment is employed at that time rather than at a second operation.

From time to time cases of tumor of the upper jaw are encountered in which a diagnosis of Vincent's infection has been

made because Borrelia vincenti was found in a smear of material from an ulcerated lesion about the jaws. It is possible to find Borrelia vincenti in almost any ulcerated lesion about the mouth; therefore, the question of acute leucemia, agranulocytic angina, lymphosarcoma or carcinoma must be considered in cases in which a diagnosis of Vincent's angina has been made. In a case of acute lymphatic leucemia, the leucocyte count alone will not aid in the diagnosis because frequently the number of leucocytes in the blood does not differ from the normal; a differential leucocyte count is necessary in order to establish the diagnosis.

Roentgenograms are of great aid in diagnosing malignant tumors of the antrum. Various types of inflammatory lesions are encountered, for example, osteitis fibrosa cystica and giant-cell tumors; also cysts, adamantinomas, fibro-osteosarcomas and various other more benign tumors are seen. All of these must be considered in the differential diagnosis of neoplasms of the antrum.

Öhngren,[5] in studying tumors of the upper jaw and antrum, has used an original classification and has included three factors: (1) the more or less favorable position of the tumor; (2) the greater or lesser degree of histologic malignancy; and (3) the presence or absence of metastasis. In charting the more or less favorable position of the tumor, which of course is a large factor in its curability, he uses a plane which passes from the inner canthus in a direction backward and downward to the angles of the mandible. In this way he separates a superior-posterior group from an inferior-anterior group. It is readily seen that, as compared with the superior-posterior tumors, those which are inferior-anterior are much more easily removable, and they may become of large size without invading regions that make their removal impossible.

TYPES OF TUMORS

In a recent review[3] of 295 cases of tumor of the upper jaw and antrum it was found that the percentage of cases of squamous cell epithelioma is much higher at present than it was twenty years ago; undoubtedly the reason for this is a change in the viewpoint of pathologists. Table 1 reveals that sixty-three of ninety-one primary tumors of the antrum were squamous cell epitheliomas and that thirty of fifty secondary tumors of the antrum were squamous cell epitheliomas. In these cases an attempt was made to distinguish the tumors which apparently had started in the antrum itself, and which I call "primary" tumors of the antrum, from those which apparently had started in the upper jaw, and which I call "secondary" tumors of the antrum. This distinction, of course, is not absolute because of the impossibility at times of determining where the tumor originated.

TREATMENT

The selection of patients for operation is based on the situation and extent of the growth, the type of tumor and the presence or absence of metastasis, and in addition, on the patient's age, general condition and ability to return for observation. These patients should be followed carefully after operation, so that if the growth recurs treatment can be instituted immediately. Recurrent adenocarcinomas and tumors of low grade of malignancy are susceptible of treatment because they are much less likely to metastasize than are some other, more highly malignant growths. Patients who have extensive, primary, antral tumors of highly malignant type have a better chance of recovery than those whose tumors are of the same size but are of a low grade of malignancy. The reason for this apparent paradox is that tumors of high grade of malignancy can be destroyed by diffuse radiation whereas every particle of a tumor of low grade of malignancy must be removed by surgical diathermy and this is very difficult to accomplish. Therefore, patients who have very extensive, squamous cell epitheliomas, of malignancy graded 4, and those who have sarcomas, are subjected to treatment.

The treatment in these cases has been described in previous reports.[2] Intratracheal gas anesthesia usually is employed and exploration is made either through the mouth or by means of lateral rhinotomy, depending on the situation of the tumor. Biopsy is performed and a fresh frozen section is examined to determine the type of growth. If lesions are of a low grade of malignancy, I feel that careful and thorough destruction with surgical diathermy at the time of the first operation offers the best chance of clearing up the disease. A protected endotherm point, with the spark gap almost closed, is used in approaching the more dangerous regions about the orbit or the ethmoid bone, whereas a large point, with the spark gap well open, is employed in regions wherein wide destruction will not be of serious consequence.

If bone is involved, the periosteum must be destroyed in order to prevent recurrence. Unless a great deal of time and care is used, some tumor will be left in place. Fresh frozen sections for microscopic examination may be made if there is any question about the presence of tumor at any point. In questionable regions radium seeds may be used. Preliminary ligation of the external carotid artery is employed only in an occasional case in which secondary bleeding necessitates its use.

In the presence of highly malignant lesions exploration should be made and enough of the tissue destroyed by means of surgical diathermy to establish very free drainage. Radium is then inserted at various points, directly into the tumor, through the wound.

Considerable reaction may follow the use of surgical diathermy and irradiation. If the growth involves the ethmoid region and considerable destruction is effected with diathermy, it is possible to cause edema of the brain, which may clear up in a few days. Meningitis is a very rare complication and is much less likely to occur in association with an ethmoid tumor if the growth is removed by means of diathermy than if surgical measures are employed. Secondary bleeding may occur. If it is not readily controlled by packing, the external carotid artery should be ligated. The sequestrum secondary to diathermy and radium is usually removed six weeks or two months after operation. About 10 per cent of patients who have tumors of the sinuses lose the eye on the side where the tumor occurred. It may be necessary to remove the eye at the time of operation or infection may necessitate its removal later. Ophthalmitis may cause loss of the eye, but not uncommonly an acute inflammatory condition of the orbit subsides, leaving vision very little impaired.

From time to time a surgeon encounters a fulminating type of malignancy of the sinuses wherein Roentgen rays or radium alone, used externally, will cause the tumor to disappear. However, this type of malignant lesion usually recurs. I do not feel that Roentgen rays or radium used alone, externally, accomplishes as much as radium inserted directly into the sinuses. If a lymph node is involved in metastasis, irradiation is used externally. I do not believe that block dissection of the neck for cervical metastasis from these tumors, unless they are of low grade of malignancy, accomplishes a great deal. I prefer to use Roentgen therapy over the neck.

In some of these cases plastic operation is required to restore the cheek or the side of

Table 1

PRIMARY AND SECONDARY TUMORS OF THE ANTRUM

Types of Primary Tumors of the Antrum	
Squamous cell epithelioma	63
Adenocarcinoma	6
Round cell sarcoma	7
Myxosarcoma	1
Fibrosarcoma and osteosarcoma	9
Lymphosarcoma	3
Malignant, type of cell not determined	2
Total	91
Types of Secondary Tumors of the Antrum	
Squamous cell epithelioma	30
Adenocarcinoma	5
Round cell sarcoma	1
Myxosarcoma	1
Fibrosarcoma	2
Malignant, type of cell not determined	11
Total	50

the nose. In such an operation on a man, I feel that tissue from the supraclavicular region, if brought up by means of a tubed flap, furnishes skin of the best color and character. If the patient is a woman, a pedicled flap from the forehead may be used to close the perforations; the resulting scarring may be covered by dressing the hair appropriately. Perforations of the upper jaw, which open into the antrum, are best closed by means of a dental plate for it is almost impossible to make a dental plate which will carry teeth and fit over a flap.

RESULTS

The operative mortality has been markedly reduced in recent years and, as compared with previous times, more patients are alive without recurrence five, ten or fifteen years following treatment. At the Mayo Clinic, recently, examinations were made of patients who had been well for fifteen and sixteen years after they had been treated for malignant growths of the upper jaw and antrum.

In a recent study[4] of 295 cases of malignant tumor of the upper jaw and antrum in which operation was performed previous to 1929, 236 patients were traced, of whom 127, or 53.8 per cent, were living without recurrence five years later. Of those who had tumors which were apparently primary in the antrum, ninety-one were operated on, seventy-five were traced and thirty, or 40 per cent, were living without recurrence five years later. Of the patients who had secondary antral tumors, fifty were operated on, forty-three were traced and twenty-three, or 53.4 per cent, were living without recurrence five years later. Of those who had tumors of the upper jaw and palate, 154 were operated on, 118 were traced and seventy-four, or 62.7 per cent, were living without recurrence five years later.

Öhngren,[6] in reporting 235 cases in which operation had been performed between 1924 and 1936, reported the end results in 120 cases in which it had been possible to follow the patients for five years or more. Of these, forty-two, or 35 per cent, were still living without recurrence from five to twelve years after commencement of treatment. These results show that malignant disease of the sinuses is curable if proper and thorough treatment is employed.

REFERENCES

1. New, G. B. Treatment of malignant tumors of the antrum. *J. A. M. A.*, 74: 1296–1300 (May 8) 1929.
2. New, G. B. End results of treatments of malignant tumors of the antrum of Highmore. *Arch. Otolaryng.*, 4: 201–214 (Sept.) 1926.
3. New, G. B. The treatment of malignant tumors of the upper jaw and antrum: fifteen years of progress. *Surg., Gynec. & Obst.*, 61: 706–707 (Nov.) 1935.
4. New, G. B., and Cabot, C. M. The curability of malignant tumors of the upper jaw and antrum. *Surg., Gynec. & Obst.*, 60: 971–977 (May) 1935.
5. Öhngren, L. G. Malignant tumors of the maxilloethmoidal region. *Acta Oto-Laryng.*, suppl. 18, 1933.
6. Öhngren, L. G. Malignant disease of the upper jaw. *J. Laryng. & Otol.*, 52: 18–27; 36–47 (Jan.) 1937.

DIETARY TREATMENT OF CHRONIC SINUSITIS

BURT R. SHURLY, M.D.

Professor of Rhinology, Laryngology and Otology, Wayne University School of Medicine

DETROIT, MICHIGAN

THE dietary treatment of chronic sinusitis is dependent for its success or value upon a definite and carefully determined diagnosis of the condition itself. Many false ideas prevail, so that it sometimes happens that a patient accepts a diagnosis of sinus disease when he has been given only a superficial examination, whereas he should have the benefit of careful observation and classification by one who is qualified to know chronic sinusitis from some of the various pathologic manifestations in the nose. However, the general practitioner is learning more about sinus pathology as he studies, reads, and takes postgraduate courses.

It is fashionable among many people today to refer to a cold as sinus disease, and to a persistent cold as a "chronic sinus." They may be right, or they may really have allergic or inflammatory conditions of the nasal mucous membrane, or perhaps the disease may be merely an affectation. Formerly we laid great stress on intumescent or hypertrophic rhinitis, and that of the atrophic variety, as a usual and more common condition of the nose. Going farther back, all nasal conditions were classified under the general name of "catarrh," and almost all people, particularly in our northern climates, believed they had catarrh—a name which covered practically all nasal pathology during the older regime.

It is, therefore advisable to understand the newer pathology, and to recognize that the chronic manifestations in the nose—other than special entities, such as syphilis, tuberculosis, rhinoscleroma, tumors malignant or benign, polypi, ozena, leprosy, and other unusual conditions—may be for the most part classified as allergic, hypertrophic, intumescent, atrophic, with definite etiology, known or unknown, which bring on the pathologic manifestations.

The more we know of chronic sinusitis, the more we understand that allergy, endocrine imbalance, infection, malformations of the cartilage or bone, with other attending obstructions, are constant factors. The more we learn of the endocrines, the thyroid, the adrenal, the ovarian glands with the estrus cycle, the pituitary located directly posterior to the sphenoid, the parathyroids, the pineal gland (of which less is known), the thymus, the liver, the pancreas with its internal secretions carrying mysteries of influence to the vasomotor mechanism—the more we learn of these, the more we are enlightened upon the interrelationship of the erectile tissues in the nose and those of other parts of the body. When we consider chronic sinusitis, and are not familiar with the vasomotor arrangements of the nose, and with the areas of lymphatic drainage, we are leaving out some of the principles which should be understood.

When pocketed pus is present, *surgery must come first*, and no dietary treatment can supplant it. We must remember that in chronic sinusitis there are almost always ten sinuses, five on each side of the nose, that can become chronically infected. Some individuals are so suspectible to infection that a series of acute colds, so-called, leave the patient a winter of subacute and chronic sinusitis. The surgical principle of drainage must never be disregarded, and no other method of treatment can take its place in certain cases. It is true that some members of the profession become so thoroughly surgery-minded, with the idea that surgery alone is of value, that they neglect many non-surgical therapeutic measures.

The consideration of a case of chronic sinusitis must be an individual investigation in every instance; we must study the patient, his resistance to infection, as well as the microorganism and its pathogenesis and possible malignancy. While recognizing fully the importance of surgery, the necessity of proper drainage, and the necessity of removing pus from the sinuses by any possible method that will eliminate focal infection, we must also take advantage of other therapeutic measures of value. Why is it that one individual succumbs to coryza, with a train of complications which exhibit a decided lack of resistance to that infection? Why is it that the next person will throw off that coryza in its first stage, and establish an immunity through a winter of continuous colds about him? These questions cannot be definitely answered, yet various theories offered have much in their favor.

For many years we were satisfied with a diagnosis of hyperesthetic rhinitis, hay fever, or hay asthma, and many hundreds of remedies were advocated for these conditions. The allergic testing, now considered routine in chronic sinusitis, reveals any irritating objects, pollens, dusts, and emanations, as well as any food sensitivity; these findings may have a direct bearing upon the symptoms, the severity, the complications, and the treatment of chronic sinusitis.

The effect of food allergies alone is sufficient evidence of the great importance of diet upon the well being of the patient. We have in the literature now a vast amount of knowledge about foods; we have come a long way from the early days of otolaryngology when horse asthma, dog dandruff, cat hair, and the yolk of eggs, milk, tomatoes, cucumbers, and strawberries were known to be disturbing and irritating to some individuals. People had to learn from experience that they could not eat strawberries without developing urticaria; now, with the allergy tests one can definitely determine an offending food, and measure it plus 1, 2, 3, or 4, according to the violence of the reaction.

Food irritations may be accompanied by swelling of the mucous membranes of the nose and vasomotor areas, and since the sinuses must have drainage from the natural openings, any swelling of the orifices can only result in a retention of pus, and an absorption of infection, harmful to the patient. The dietary treatment of chronic sinusitis, therefore, entails first, an allergy test.

The metabolism test is also important to determine the status of thyroid function. We often find that persons with food allergy have some form of endocrine imbalance, as well. The two conditions must be corrected as far as possible. The great value of small doses of thyroid and Lugol's solution, as indicated, should be noted here.

After the study of food sensitization, the dietary treatment of chronic sinusitis can be utilized to better advantage. When irritating foods are eliminated, the foods with a proper supply of vitamins can influence the structure of the nose, throat and ear in a very remarkable way. In our own research laboratory, after a year's work, Dr. R. G. Turner and I have demonstrated infection of the accessory sinuses and upper respiratory tract in white rats and in monkeys as a result of vitamin A deficiency. Vitamin A is essential to the animal and to the human; without it, or with even a slightly diminished supply, a definite line of symptoms develops, with respiratory infection, ulceration of the cornea, ulcers at the base of the tongue, drying of the lacrimal apparatus, and lowered resistance to infection. In our series of observations, the Staphylococcus albus, the Friedlander-like bacillus, and the chromogenic group 6, were active. With the continuation of a diet deficient in vitamin A, white rats developed suppuration of the sinuses and, in many instances, suppurative otitis media. With the resumption of an adequate diet containing vitamins A and

D, these symptoms cleared up in from two to three weeks.

If we look for good resistance to infection, it must come through the blood stream and lymphatics, and is dependent therefore upon the digestion and assimilation of good food and upon cell selection. Many years ago, when our knowledge of dietetics was rather hazy, we felt a need for something to efface malnutrition, to build up better metabolism after tonsillectomies, and to assist thin and anemic patients. At that time, some forty years ago, I was fortunate enough to hit upon a tonic, which came to be called Shurly Soup. It contained two carrots, two tomatoes, two beets, two potatoes, and a bunch of celery; these were ground up in a meat press and strained to remove the bulk. The raw juice was served cold, a wine glass full to a child 10 years of age, three times a day. Seasoning extracts or malt could be added, or anything else that would make it more palatable. This proved to be a valuable adjunct to treatment, especially for growing children. In those days we gave syrup of iodide of iron, or the syrup of hypophosphites; medicine was considered of first importance, and food second. Now we believe the order should be reversed. We have found that the diet of the individual with low vitality, low resistance, chronic suppurative diseases in the sinuses and the ears, demands an adequate vitamin supply. We can make no mistake in adding to all other therapeutic measures the dietary treatment.

In this connection, I want to mention the fish oils, which have a decided value toward the nourishment, stability, and health of the tissues of the ear, nose and throat. Cod liver oil is valuable, and halibut liver oil (which has a much greater strength, one drop being equal to three teaspoonfuls of the cod liver oil) is even better. There is danger, however, that the excessive use of cod liver oil may produce an intoxication. Steck and his associates have reviewed the problem in the Annals of Internal Medicine (January 1937) and show that hypervitaminosis D may produce symptoms of hyperparathyroidism. Both human subjects and dogs generally survive the daily administration of 20,000 units per kilogram for indefinite periods without intoxication.

In addition to the fish oils, eggs, milk, leafy vegetables and carotene are very helpful; also cracked wheat, which contains Vitamin E in quantity.

My interest in the nutritional problems related to our speciality was greatly stimulated by a case of scurvy that entered Shurly Hospital some years ago. The patient exhibited the factors of faulty diet, weakness, mental depression, hemorrhage from the gums, ears and nose, with purpura and subperiosteal hemorrhage and tenderness. The diagnosis was confirmed by a series of blood counts. There was rapid recovery with the feeding of iron, calcium and vegetables, including the anti-scorbutic vitamin C, in oranges, lemons, limes, tomatoes, and grapefruit. The vitamin C foods, plus vitamin D in red bone marrow, and a tonic rich in calcium and containing malt, pulverized ossis, lactic acid, pancreatin, and pepsin, were continued for some months. Two years later, the patient was entirely well and has had no relapse.

We are beginning to appreciate the value of the raw mineral salts in sinus disease and other infections. More than seventeen chemical elements are required in the chemistry of the cell. We have known for a long time the value of normal salt solution in washing out sinuses, and the importance of maintaining six-tenths of 1 per cent of normal salt in the blood stream. We have witnessed, too, the merit of potassium salts in raw vegetable juices. Iodine is essential, as the goiter zones of Switzerland and northern Michigan well demonstrate. The prevalence of chronic sinusitis in the lowered resistance to infection as exemplified by hyperthyroid cases is well known. On the importance of calcium in its relation to sinus infection, as produced by the parathyroid glands and the pineal, we require further elucidation. Lowered resistance from chilling of the sinuses and the

disturbance of the vasomotor nervous system, is in direct relation to proper diet in the supply of hot, high vitamin content food. We are awaiting with interest further experimentation and clinical proof regarding vitamin K, as found in alfalfa, which seems to have a bearing upon hemorrhages from the mucous membranes of the nose and sinuses and other parts of the body. We know well that the bony formations of the sinuses are greatly dependent upon a proper supply of vitamin D, in the prevention of rickets. In fact, the bony frame of the nose may be altered in early childhood by the right supply of cod liver oil, or its equivalent. The ratio of calcium and phosphorus in the formation of the sinuses and the size of the natural openings is an important factor in the development of a chronic sinusitis.

The question of decay of the teeth and abscess formation in relation to the floor of the antrum, is essentially a dietary problem. The presence of small jaws and crowded teeth, with enlarged tonsils and adenoids, belongs with faulty skeletal development and is influenced by dietary errors that favor rickets. Malposition also favors food packs and foci of fermentation. Diet is particularly important in children, as many of these sinus cases become chronic very early in life, especially those with an antrum involvement. Spinach and carrots are good foods. The milk must be above 3.5 fat content, as the formation of the sinuses is influenced by mother's milk of proper constituency, or the right formula for the individual child.

Research in the field of nutrition applied to preventive otolaryngology offers an opportunity to raise the vitality of all mankind. The errors of diet may not cause deficiency disease, but insidiously their gradual operation, more or less constant, may be responsible for undermining the resistance in a degree so masked that the menace is quite unappreciated. As a prophylactic to chronic sinusitis I would therefore stress dietetic treatment, including especially carotene, cod liver oil or one of the fish oils, milk and cream, spinach, carrots, with a good supply of mineral salt, orange juice, and grapefruit juice.

CONCLUSIONS

The treatment of chronic sinusitis calls for surgical intervention. Dietetic treatment should be used as a therapeutic aid.

The prophylactic treatment in early infancy may offer much in preventing bony malformations of the nose and sinuses, by a proper use of vitamin D, particularly in milk, eggs, and leafy vegetables.

Vitamin A deficiency may appear as a borderline manifestation and influence the resistance to infection, with lowered vitality of tissues, especially in the ear, nose and throat.

Vitamin C, as a preventive of scurvy, has a special influence on the mucous membranes of the gums, nose and mouth.

The value of raw vegetable juice and mineral salts lies in promoting cell growth and selection.

Special attention to endocrine balance with the use of iodine and thyroid extracts is worthwhile in establishing vasomotor tones.

Food allergy must be determined by special tests and history. The prevention of sinus infection at the time of the removal of tonsils and adenoids, may require special dietetic treatment and cod liver oil.

More than 20,000 units of vitamin D per kilogram of cod liver oil may result in intoxication with symptoms of hyperparathyroidism.

In a case of chronic sinusitis, after necessary surgery special inquiry should be made regarding the diet of the individual, so that the physician may give intelligent advice toward increasing resistance to infection.

ON THE APPLICATION OF ZINC SULFATE SOLUTION IN THE PREVENTION OF POLIOMYELITIS

EDWIN W. SCHULTZ, M.D.

Professor of Bacteriology and Experimental Pathology, Stanford University School of Medicine

STANFORD UNIVERSITY, CALIFORNIA

ALTHOUGH the protective action of zinc sulfate solution in the prevention of experimental poliomyelitis has been firmly established,[1] it is not yet possible to say that it is effective in man, nor possible to prescribe the exact procedure which should be followed in applying the prophylactic treatment in man. Indeed, knowledge of the practical usefulness of this measure is still so meager that almost anything one may say at this time is likely to require early revision. This paper should, therefore, be regarded as a review of the present status of a very new development in preventive medicine.

In the face of a striking laboratory result uncertainty as to the practical usefulness of a measure may be difficult to understand. However, in this instance there are several questions which must be definitely answered before one can discuss with understanding the practical value and limitation of the measure. In the first place, while it seems fairly certain it is not yet definitely established that in man the virus of poliomyelitis always enters by way of the olfactory nerve.[2] Even granting that it does enter solely by this route, there is no justification for being confident that the results obtained in monkeys will apply to man. Furthermore, while the risk of inducing a permanent anosmia by the use of zinc sulfate is recognized,[3] we do not yet know the extent to which this risk may be eliminated without sacrificing the effectiveness of the measure. Unfortunately, the gap separating the discovery of a measure effective in experimental animals and the establishment of its practical usefulness in man may be a very wide one. In the case of zinc sulfate prevention of poliomyelitis, we are still quite definitely engaged in an endeavor to bridge this gap.

In this brief summary of the problem no attempt will be made to present the details of the work which led up to the experimental trial of chemicals in the control of this disease. Such reviews have already been published.[2,4] It will probably be sufficient to state that resort to chemical prophylaxis was the outgrowth of observations showing that the infection in this disease is primarily neuronal and that serums and vaccines are ineffective in controlling it. Studies of its pathogenesis, including investigations on the cytologic changes induced, have revealed that the virus is transmitted axonally, and from the beginning is probably in an intracellular relationship. An appreciation of these facts is fundamental to an understanding of the disease and of the problems which it presents. The relationship, for example, explains why the use of immune serums, prophylactically as well as therapeutically, has proved disappointing. In this the situation is analogous to that of a cat trying to reach a mouse in a long small-sized tube, the opening of which is in the superior common nasal meatus. Immune serum may bathe the nerve cells, but the neutralizing substance in the serum being a relatively large aggregate, it is unable to enter the cell and contact the virus inside. Only such virus as may happen to leave the neuron can be contacted by the immune substance. The open end of our hypothetical tube, namely the olfactory nerve endings, are probably not effectively bathed with immune substances derived from the blood plasma and therefore no

efficient barrier is set up at the portal of entry.

Present information of its pathogenesis and of the basis of acquired immunity affords an explanation as to why vaccines, even those containing living virus, have proved relatively ineffective in preventing the disease. It is now known that the injection of vaccines accomplishes no more than a stimulation of humoral antibody production, but as I have already stated, humoral antibodies are relatively ineffective either in preventing infection or in arresting its progress once established. In this disease acquired immunity does not rest to any important degree on humoral antibodies, but rather more on some change in the protoplasm of susceptible nerve cells induced as the result of intraneural contact with virus. However, no way has yet been found for inducing such a cell-modifying infection by artificial means without a parallel risk of inducing a paralytic form of the disease. It is possible that such a method of prevention may eventually be found, although there is now no evidence to suggest that a practical solution of this kind will become available soon.

It was the failure of orthodox immunologic procedures which more than anything else led to attempts to control the disease by chemical means. In view of the evidence that the virus is highly selective in choosing its pathway to the central nervous system and seems to do so only by the way of the olfactory nerve endings, the possibility of modifying the permeability of this portal offered an attractive approach. Subsequent investigations have sustained the hope that a practical solution may lie in this direction. A number of agents have been found to afford protection by such direct action on the olfactory portal. Among those which afford more or less protection are: alum, mercurochrome, picric acid, tannic acid, and certain water soluble zinc compounds.

We have found zinc sulfate outstandingly protective among some fifty substances tested. A single treatment with 1 per cent zinc sulfate affords complete protection to about 90 per cent of monkeys against virus instilled intranasally a month later. The protection may last longer than a month, but the resistance tends to drop off gradually until eventually most of the animals are again susceptible.* Lower concentrations of zinc sulfate are, of course, less effective, but a single application of 0.5 per cent solution may protect more than half the animals against virus administered a month later. With concentrations of less than 1 per cent several successive daily applications of the solution may be necessary for uniform protection. Five successive daily applications of a 0.25 per cent solution have proved as effective as a single application of a 1 per cent solution. A full report of our experimental observations will be published later. It is not essential for this discussion to present them in detail, for as I shall point out later in this paper it is quite possible that in the practical application of the prophylactic treatments in man, the treatments need not parallel those in monkeys quite as much as we previously supposed.[4] Because of the risk of inducing a permanent anosmia,[3] it is necessary to approach rather cautiously the concentration of the drug which may be safely applied in man. This is, in fact, what I want to discuss particularly.

To understand the practical implications of this type of prophylaxis, we should give thought to the possible mechanism underlying the protection afforded by zinc sulfate solution in monkeys. This in turn calls for a brief review of the anatomy of the olfactory apparatus.

In man, the olfactory mucosa occupies the upper third of the nasal septum, nearly the whole of the superior concha and a small portion of the middle concha (Schaeffer[5]). In the healthy and fresh state the

* This reversion to a susceptible state is of considerable practical importance, not only from the standpoint of the temporary character of the resistance, but, as I shall point out later, in connection with the question of the extent of the olfactory impairment necessary for protection and the question of the permanency of the impairment.

area is demarcated from the adjacent mucosa by its yellowish or brownish tint and slightly greater thickness. Histologically, the membrane presents three kinds of cells: (1) olfactory cells, (2) sustentacular or supporting cells, and (3) stellate basal cells. These cells rest directly on the lamina propria, there being no intervening basement membrane. Among these the olfactory cell is, from our standpoint, by far the most important, while the sustentacular and basal cells are the most conspicuous and easily identified.

The sustentacular cells are tall and columnar, with fairly large, vesicular, oval nuclei. The superficially lying portion of the sustentacular cell extends a considerable distance peripherally to the nucleus, and together with similar portions of other cells gives rise to a superficial zone in the olfactory mucosa which is practically devoid of nuclei. The cell is not ciliated. Its deep lying portion is said to be forked, the processes ramifying with those of neighboring cells to form a network.

The basal cells make up the lower stratum. They may be either pyramidal, cuboidal or rounded in shape and present oval nuclei resembling those of the sustentacular cells.

The olfactory cells are fusiform bipolar cells which present a large spherical nucleus surrounded by a narrow zone of finely granular protoplasm. Their nuclei are larger and rounder than those of the cells already described and lie for the most part between two zones of oval nuclei—those of the sustentacular cells and those of the basal cells. As is well known, this cell has two processes. The superficial process, or dendrite, is short and relatively thick, its actual length depending on the level of the cell in the mucosa. It courses through the non-nuclear zone and terminates slightly beyond the free surfaces of the epithelium in a small hemispherical or bulbous enlargement. This is surmounted by six or eight short cilia-like olfactory hairs, which project often spine-like from different parts of the rounded end. Occasionally the dendrite of the olfactory cell does not possess this bulbous enlargement, but itself projects spine-like and terminates in one or more hairs. These details are, of course, not fully revealed in routinely stained sections. To bring them out, special staining is necessary. It is largely because the olfactory cells and their processes are difficult to demonstrate with certainty that certain practical questions remain unanswered.

The deep process of the olfactory cell is thread-like from the beginning and is continued centrally as an axon. The axons from neighboring cells run together to form eventually about twenty nerve filaments which pass through as many openings in the cribiform plate to end finally in the glomerulus of the olfactory bulb. The nerve fibers of the olfactory nerve are unmyelinated and in routinely stained preparations their course is recognized by the characteristic nuclei of the Schwann cells. Each of the main filaments or branches of the nerve receives a tubular sheath from the dura and the piarachnoid, the former being continued into the periosteum, the latter into the neurolemma of the nerve. In the olfactory glomerulus the axons form a synapse with the dendrites of the mitral cells of the olfactory bulb. The axons of the latter cells pass by the way of the olfactory tracts to the brain. There is no reason for bringing these into the present discussion, except to make way for the statement that after the olfactory tracts have been sectioned in monkeys virus can be instilled intranasally repeatedly without inducing infection. This shows that the portion of the nasal mucosa through which infection readily occurs is limited to the olfactory region.

With evidence that the olfactory nerve serves as the portal of entry, we may proceed a step farther and attempt to localize the ultimate portal. We have noted that the bulbous end of the olfactory dendrite, as well as the olfactory hairs surmounting it, protrudes beyond the general surface of the mucosa. Since the virus of poliomyelitis is among the smallest of the known

filter passing viruses (about 10 millimicra[4]), it may conceivably invade directly the exposed bulbous dendrite, and even the delicate olfactory hairs surmounting it. Whether it does so at one or the other of these exposed parts of the nerve may be relatively unimportant. What is of importance is that the terminals of this nerve are exposed to the exterior and therefore easily accessible to the virus. While it is entirely possible that the virus may invade the deeper lying olfactory cell body proper, this would appear less likely than a direct invasion of dendritic processes.

If the ultimate portal of entry for this virus is the exposed portion of the olfactory nerve, one may then be permitted to indulge in some speculation as to how a protective chemical might act in inducing the resistant state. Theoretically, a state of resistance might result from a chemical action limited to the olfactory hairs and the exposed bulbous portion of the dendrite. The chemical effect might result either from a nondestructive union of the chemical with the nerve endings, simply modifying the permeability of the protoplasm[2,6] or from an outright destruction of these terminals. In the first instance susceptibility to the virus would not recur until a reversal of the chemical combination had occurred, in the latter it would not return until a sufficient number of receptors had undergone regeneration. Of course, protection may result also from a destruction of the olfactory cell bodies themselves, along with other epithelial cells in the mucosa. In the latter case a return of susceptibility would not be expected, unless Nature has provided an exception in the case of olfactory neurons with regard to regeneration of nervous tissue. There are certain reasons, which we cannot discuss here, for not excluding altogether the possibility that destroyed olfactory nerve cells may be replaced in some way.

These are the theoretical possibilities. We may ask now what do microscopic sections of the olfactory mucosae of treated animals show? Abstractly there would appear to be little difficulty in determining which of the above mechanisms underlies the protection. Actually, however, considerable difficulty has been experienced, chiefly because of the lack of altogether practical methods for differentially staining the olfactory cells and their processes. For this reason it is not yet possible to speak definitely of the degree of olfactory impairment necessary for protection and therefore not possible to estimate the practical limitations of this type of prophylaxis. Until a fuller understanding of the degree of the involvement necessary is reached, one must speak with reservation. Should it prove eventually that for effective protection the involvement must extend to the olfactory cells themselves, it would then be quite obvious that such a prophylactic measure could have no practical application in man. If, on the other hand, a high degree of protection may result from a less drastic action limited to the dendritic endings of the nerve, there would then be reason for continued hope that the measure will prove of practical value.

In a preliminary report of our observations on the experimental application of zinc sulfate[4] we stated that we had "not observed any noteworthy gross or microscopic changes in the nasal mucous membranes of monkeys treated repeatedly with zinc sulfate other than a mild grade of inflammation, such as may be seen at times in untreated animals." This statement was based on membranes removed several days to several weeks after treatment. Since then we have examined many more specimens, including membranes removed less than one day to more than four months after treatment with varying concentrations of zinc sulfate solution.* It is true

* Most of these have been removed in toto with the olfactory nerve filaments and olfactory bulbs attached. If properly oriented in the process of embedding and sectioned tangentally, such material affords an opportunity to study the various structures essentially in their normal relationships. In the absence of an altogether satisfactory special method for staining the olfactory cells most of the sections have been stained by routine methods. While material so stained is not entirely satisfactory from the standpoint of deter-

that mucosae removed some days after treatment and stained by routine methods may show little apparent deviation from the normal structure, especially if one takes into consideration the variations in structure noted in untreated animals. It was something of a surprise to us to note the variations in the structure of the olfactory epithelium in normal monkeys; only in some does the histologic picture fit fully descriptions of normal olfactory epithelium. What surprised us especially was the remarkable regenerative powers exhibited by the olfactory epithelium, so that a severely damaged mucosa may within the course of a week or so present a structural appearance which, superficially at least, is not very unlike that seen in some untreated animals.

To obtain a full picture of the effects produced by zinc sulfate solution one must remove the treated membrane within twelve to twenty-four hours. Membranes removed this early generally show a more or less marked seropurulent exudation into the meatus, edema and PMN infiltration of the epithelium and the lamina propria, and what is of especial importance, more or less necrosis and desquamation of the epithelium.

Damage to the epithelium is likely to be especially extensive following treatments with 1.5 per cent* solution of zinc sulfate. With this concentration the damage is so extensive that much of the olfactory epithelium, down to the layer of basal cells, or to the lamina propria may be stripped and lie in folded shreds or ribbons within the meatus. The nuclei of the exfoliated epithelial cells are usually well preserved, but the cytoplasm of the cells generally shows a change in staining properties.† In some areas, the killed epithelium may not yet have separated from the underlying living tissue. A separation of the epithelium down to the layer of basal cells or the lamina propria or a necrosis extending to this depth means, of course, that in these areas at any rate the olfactory cells have been included in the epithelial necrosis.

Lower concentrations of zinc sulfate are naturally less damaging than the stronger solutions although extensive epithelial necrosis may be observed following a thorough application of a 1 per cent solution and even following treatments with 0.5 per cent solutions. The damage is usually much more marked in the upper portion of the olfactory region where the surfaces lie close together. Even a 0.1 per cent solution may produce some necrosis in this region if the solution is thoroughly applied and allowed to act for a sufficient length of time.

It is a noteworthy fact that the damage to the epithelium is always greater in the superior portion of the olfactory area, while the lower portion and especially the respiratory epithelium rarely shows more than an acute inflammatory exudation. It seems certain that the olfactory epithelium is very much more susceptible to chemical trauma than is the respiratory epithelium. It is, however, also possible that the lesser damage to the respiratory epithelium depends in part on freer drainage, and, therefore, on a shorter period of drug action.

No doubt an important factor in the prophylactic effectiveness of zinc sulfate solution in monkeys is to be found in the structure of the olfactory region. In the monkey the superior common nasal meatus is an extremely narrow cleft, in which the surfaces of the olfactory mucosa are separated by only a fraction of a millimeter.

mining the fate of the olfactory cells, considerable information of practical importance can be obtained from such sections.

* While this is a stronger solution than we have found necessary to obtain uniform protection in monkeys, we have employed concentrations as high as this in these histologic studies partly on the assumption that mucosae more than usually sensitive to this drug might be similarly affected by lower concentrations.

† The reason for this probably rests on the fact that zinc sulfate solution in such concentrations is a good tissue fixative. Since this is the case, any separation of an apparently intact strip of epithelium should be regarded with suspicion and not necessarily as the probable result of some mechanical action (manipulation in removing the membrane, tearing by the microtome knife, etc.).

This cleft extends about 1 cm. anteroposteriorly and about 0.5 cm. vertically. A space so narrow and of this area would tend to retain fluid by capillary attraction. Naturally, any tendency for the drug to be retained in this space would lengthen the time that the chemical can act. The longer the action the more deep seated would the effect on the epithelial cells be likely to be. It may, therefore, well be that the remarkable protection observed in monkeys is determined in large part by the effect of a relatively prolonged drug action. To determine the possible influence of this factor an experiment is now in progress in which one series of monkeys has been treated with zinc sulfate solution in the usual manner; while in another series the application of the zinc solution has been followed in about one minute with an irrigation of physiologic saline to check the duration of drug action. The results of this experiment should prove of great practical interest.

So far as we are aware, lasting anosmias in man following the application of zinc sulfate solution have occurred only in adults. According to our information, the sense of smell in children has practically always returned to normal within a period of about two weeks. In adults, however, olfactory function may be absent for as long as four months and we have been informed of five cases in which the sense had not returned after more than six months. Not only does the anosmia tend to be more lasting in adults, but we have been informed that grown persons tend to experience a more painful post-treatment reaction than do children. This reaction generally takes the form of a severe headache, which usually begins about twenty minutes after the treatment and lasts from three to six hours. It is usually a frontal headache, localized between the eyes and referred to the occiput; it often terminates suddenly, "as though a hot iron had suddenly been removed."

The tendency for adults to suffer a more severe post-treatment reaction and a longer anosmia calls for some explanation. To us it seems that the difference may rest on anatomic differences influencing the length of time that the solution may remain in contact with the olfactory mucosa. From observations on human cadavers, as well as from the fact that a smaller volume of fluid is required to fill the olfactory space (vide infra) it seems evident that the superior common nasal meatus in adults is actually, as well as relatively, much narrower than in children. In some of the cadavers examined by us the width of this space appeared to be less than a millimeter. A space no wider than this, especially if sufficiently long and deep, could conceivably hold solutions by capillary attraction for quite some time. As previously stated, any tendency for the drug to remain in this space would prolong its action and increase its destructive potentialities. If this explanation is correct, then the practical implications are obvious. It must be met by efforts to control the duration of the drug action. This may possibly be accomplished fully enough by washing the drug out of the superior common meatus with physiologic saline, perhaps a minute or two after instillation. The technique for this will be discussed later.

Granting that it is possible to avoid the induction of a lasting anosmia by limiting the concentration of the drug and by checking the duration of its action, there are still other considerations which concern us in connection with the practical value of such a prophylactic measure. Most important among these is the uncertainty that zinc sulfate in safe enough concentrations will actually prove protective for man. About this we are still in complete ignorance. Although two field tests have been carried out thus far, one with picric acid-alum solutions during the summer of 1936 in Alabama[7] and one with zinc sulfate during the summer of 1937 in Toronto and elsewhere, neither of these has thrown any light on the possible effectiveness of chemical measures in controlling the disease in man. The reason for our failure to obtain

this information is found in the defective methods employed in applying the solutions, for in neither of these tests was there assurance that the technique employed sufficed to insure coverage of the olfactory area with the solution. During the Alabama outbreak the treatments were carried out with ordinary atomizers and the application of these fell largely into the hands of the laity.[7] We are fully aware now that such atomizers do not in any sense assure coverage of the olfactory area with solution.

To overcome the interference offered by the middle turbinate, Peet, Echols and Richter[8] at the University of Michigan designed a special atomizer tip, which can be introduced along the nasal septum past this turbinate. This was the method employed during the Toronto epidemic.[9] While apparently fairly efficient in the hands of Peet and his associates, and of Hayden[10] and others, working with small groups, in the Toronto field test in which large numbers were sprayed the method fell far short of the hopes entertained for it. Its application induced a temporary anosmia in less than 14 per cent of those who received one treatment and in not more than 24 per cent of those who were treated twice on two separate occasions. Since the observations of Echols, Richter and Peet,[11] Pentecost,[12] and Hayden[10] show that a temporary anosmia always follows complete coverage of the olfactory area with a 1 per cent solution of zinc sulfate, it is plain that the vast majority of those treated during the Toronto epidemic failed to receive complete coverage of the olfactory area. It is obvious that the prophylactic efficacy of any chemical agent cannot be judged unless full coverage of the olfactory area with the solution is assured. No statistical difference in the incidence of poliomyelitis in the treated and untreated groups was observed in the Toronto field test.[9] How much this can be attributed to imperfect application or to the absence of a continued effect cannot be estimated.

I have referred to the defects inherent in the method devised by Peet and his associates.[8] While the method helped to overcome an important obstacle in the treatment of the olfactory area by the spray method, it is difficult to apply and hazardous, especially in young children who do not coöperate fully. Its almost complete failure in the Toronto epidemic is testimony enough that it failed to solve the important problem of finding both an effective and an easy way of covering the olfactory area with solution. Granting its effectiveness when skillfully applied, other things militated against its becoming a practical public health method. Its application called for special training and skill and probably not enough rhinologists would be available in any given community to give all the treatments that might be required during an emergency. Thanks to the splendid interest shown by rhinologists the difficult spray method has been displaced by an easily applied and more effective postural method.

During the past summer, Pentecost[12] at Toronto, anticipating that he might be called upon to apply zinc sulfate, undertook experiments to determine the best method for its application. He finally decided on a technique which consisted of introducing the solution with the aid of a flexible olive-tipped ureteral catheter. This was inserted between the nasal septum and the middle turbinate; then with a Luer syringe attached to the free end, 0.5 c.c. of the solution was forced through the catheter. The patient was recumbent with the head in the Proetz position ("head inverted so that the chin and external auditory meatuses are in the same vertical plane"). This procedure seemed easiest to carry out, least hazardous and least discomforting to the patient. Of special importance is the fact that the method induced an immediate anosmia in 100 per cent of those treated. There can be no better testimony to its effectiveness in covering the olfactory area. Approximately 100 children, eight years of age and older, were treated by this method without serious after-effects. In February (1938) Dr. Pentecost kindly informed me that all

patients so treated had regained the sense of smell, although in a few it was still not quite back to normal. There were no cases of poliomyelitis in the treated group, but the number treated was far too small for this to be of much significance.

Early in the summer of 1937, Shahinian, Bacher, McNaught and Newell[3a,13] in San Francisco undertook similar studies to determine the best way to apply solutions to the olfactory area. Soon after beginning their work, they demonstrated a method similar to that described later by Pentecost. The method was, however, abandoned when they found that a correct posture alone overcomes the chief difficulty. They discovered that when the head is completely inverted (extended more than in the Proetz position) so that a line between the infraorbital ridge and external auditory meatus is in the horizontal plane, limited quantities of fluid, if introduced slowly into the olfactory sulcus at the limen nasi, will flow along the nasal groove and fill the inverted common nasal meatus in a selective manner and to a height usually sufficient to immerse most of the estimated olfactory area. In carrying out the procedure the patient is put on a table with the head in the fully inverted position as indicated above. A maximum of 0.5 c.c. of the fluid is introduced slowly and steadily, drop by drop, with a medicine dropper, after which the patient is kept in the inverted position for one minute. He is then turned over to the prone position and instructed to lift his head and sniff outwardly to help discharge excess fluid.

We would modify the Shahinian procedure to the extent that we would repeat it at once and in its entirety, in the repetition using physiologic saline solution; the objective of this is to dilute and remove any of the zinc solution that might tend to remain in the superior common nasal meatus by capillary attraction. Such a washing might in fact be repeated several times, especially in adults and in others whose facial contours are such as to suggest the presence of an unusually narrow superior common nasal meatus.

Shahinian and his associates believe that preliminary shrinkage of the mucous membranes is unnecessary and probably undesirable.

In the May issue of the *Bulletin of the American Academy of Ophthalmology and Otolaryngology*, Furstenberg and Hauser[14] at Ann Arbor have suggested a procedure which differs from the Shahinian method only in certain minor respects. These investigators suggest beginning the treatment by first spraying the nasal passages with 1 per cent ephedrine in saline solution, about two minutes before the patient is placed in the dorsorecumbent position. This, it will be recalled, is considered unnecessary and is advised against by Shahinian and his associates. Furthermore, Furstenberg and Hauser suggest the instillation of 1 c.c. of solution into each nostril, which is about twice the volume suggested by Shahinian. Inasmuch as the latter found that 0.25 c.c. suffices to fill the inverted superior common nasal meatus in adults, and that not more than twice this amount suffices to do so in children, it would seem that 1 c.c. is much more than is necessary to immerse the olfactory area. In view of the desirability of avoiding a spilling over of solution into the accessory sinuses, and thereby reducing post-treatment discomfort, etc., it would seem desirable to investigate further the average volume required to just cover the olfactory area, preferably for different age groups; also to investigate further whether the nasal mucous membranes should be subjected to previous shrinkage.

On the optimal position of the head the two groups of investigators are in agreement. Furstenberg and Hauser express it as hyperextension of 15 degrees beyond the Proetz position.

The postural method undoubtedly fulfills the need for a relatively simple and effective method of covering the olfactory mucosa with solution. The most difficult question to answer at this moment is

whether adequate treatment with zinc sulfate solution can be actually free enough from the risk of inducing a lasting anosmia to justify extensive application in the field. I have already pointed out the histologic changes in the mucosae of monkeys treated with various concentrations of zinc sulfate solution. These observations, along with the experience thus far obtained in man, point clearly to the existence of such a risk. But while the histologic effect on the olfactory mucosa in monkeys tends to be rather marked, especially when higher concentrations of the chemical are used, it should be borne in mind that not only were these animals very thoroughly treated, but that the anatomy of the superior common nasal meatus in monkeys is such as to favor greatly retention of the solution and prolonged drug action.

The most important question at the moment is, can the action of the drug in actual practice be controlled sufficiently to make the risk of permanent anosmia negligible and yet provide protection? Theoretically this should be possible. It now seems entirely possible that in suggesting the use of a 1 per cent solution of zinc sulfate in man (the concentration which had proved most effective in monkeys) we were somewhat in error. It seems more than likely to us now that the concentration of the drug which should be used in man needs to be determined by some preliminary tests in man, rather than based so largely on the concentration found most effective in experimental animals.

The procedure in determining the concentration to use in man should prove simple and harmless enough if approached cautiously. The chief guide in carrying out such an investigation is the sense of smell, the objective being to determine the least drug action necessary to modify or impair the endings and only the endings of the olfactory nerve. With such an objective in mind the problem becomes one of determining the lowest concentration of the drug, which, when applied by an accepted procedure (e.g., the Shahinian method), will just produce anosmia. With such an approach in mind, one would begin the treatments with, say, a solution containing only 0.1 per cent zinc sulfate, U.S.P.* (instead of 1 per cent solution, the concentration found almost uniformly protective in monkeys). Even with such a low concentration of the drug, the duration of its action should be controlled by following the treatment within about one minute with an instillation of physiologic saline (about 1 c.c.). In carrying out such tests, the sense of smell should be measured by some dependable method (the Elsberg method[15]), both before and immediately after treatments, and the subject asked to return for a further test five or six days later. In the event an anosmia is produced by this concentration (0.1 per cent) and is still present when the subject returns, no further treatments should be given. He should, however, be asked to return for further functional tests at intervals of about five days. In the event the sense of smell was not impaired by a 0.1 per cent solution the treatments may then be repeated with a 0.2 per cent solution and so on through a 0.3 per cent, 0.4 per cent to a 1 per cent solution, if this latter concentration eventually proves necessary. Should the functional test after a given treatment fail to show immediate impairment, I would not repeat the treatment at once, but wait several days, repeat the functional test, and then give another treatment with the next higher concentration, if necessary.

The above approach to the correct concentration of the drug to use in man would be purely in the nature of a preliminary investigation. Obviously, it would be desirable to have this information in hand before an epidemic makes its appearance so that an *average minimal anosmic dose* might be specified in the face of the epidemic. How-

* It should be remembered that C.P. zinc sulfate contains only two molecules of water of hydration, while the U.S.P. product contains seven. A 1 per cent solution of the C.P. product is therefore the equivalent of a 1.4 per cent solution of the U.S.P. preparation. The U.S.P. product is here referred to in specifying concentrations of the drug.

ever, should such information be lacking at the time of an outbreak of the disease it would seem wisest to "work up" to this dose, even at some risk of not producing immediate protection, rather than use a concentration higher than necessary to just produce anosmia. In such cases a record should be kept of the functional effect following each treatment, and a state of resistance should not be regarded as having been induced until anosmia is present. The experiences of individual physicians, as well as of groups of investigators, should be made available for future guidance as soon as possible.

In applying the drug the Shahinian method should probably be followed, unless some modification of the method is officially adopted by rhinologists in the meantime. What is, I am sure, of especial importance in giving the treatments, is that the zinc sulfate instillations should be followed immediately by at least one or two instillations of physiologic saline, applied preferably by the same method. It is evident that little information can be gained regarding the practical limitations of this drug unless the duration of its action on the tissue is brought under control. And since adults are likely to suffer a more severe posttreatment reaction and are in somewhat greater danger of developing a permanent anosmia, special care must be exercised in the treatment of these to keep the action of the drug under control.

The solution of zinc sulfate used should probably contain from 0.5 to 0.8 per cent NaCl, depending on the amount of zinc sulfate present, on the theory that the more nearly the solution approaches isotonicity the less irritating it may be to the mucous membranes. Local anesthetics should probably not be included in the solution, especially if the solution is to be applied by the postural method, for the reason that the tracheal reflex may be abolished by the anesthetic should any excess fluid trickle into the pharynx. We have found that monkeys treated with a zinc sulfate-pontocaine solution while being held in the dorsorecumbent position, are very apt to die of pneumonia a day or two later. This has not occurred in animals treated with the same mixture while being held in a fully inverted position in special animal holders. Although there may be less risk of such a complication in man, this possibility should, nevertheless, be kept in mind, especially if a volume of solution larger than necessary to cover the area is instilled by the postural method.

I have emphasized the importance of anosmia as an index of complete coverage of the olfactory mucosa. Theoretically at least this should serve also as a guide to the necessity for further treatments during the period of an epidemic. It seems probable that with return of the sense of smell there is also a return of susceptibility to infection, for a return of function presumably can occur only with an anatomic restoration of the portal of entry. Therefore, unless the child is kept anosmic for the entire period of the epidemic there can be no assurance whatever that it will continue to be protected against the disease.

Although the risk of inducing a lasting anosmia is an important concern at the moment, out of the thousands treated with zinc sulfate solution during the summer of 1937 only five such cases have become known to us.* It is, however, also true that the spray method used was much less effective in covering the olfactory area

* Since this paper was written Tisdall et al.[16] have reported fifty-two cases showing disturbances after six months. Seven of these showed disturbance of smell only, eight of taste only and thirty-seven of both smell and taste. The report is based on information which the authors received from various otolaryngologists who administered the zinc sulfate spray during the 1937 Toronto outbreak, the total number sprayed probably not exceeding 5,000. While these observations are undoubtedly of great practical importance, it is unfortunate that the functional status of these special senses before the sprays were given is not known, particularly since it is not entirely clear why disturbance of the sense of taste should have resulted, especially of *taste alone*. One suspects that in these persons the sense of taste may not have been well developed, a deficiency which is not uncommon. It is known, of course, that taste in the broad sense is influenced by the sense of smell and to the extent that there is impairment of the latter there would be impairment of the former.

with the solution than is likely to be the case by the postural method. It is apparent that the more efficient the method of applying the zinc solution the greater will be the risk of inducing a lasting anosmia.* This increased risk may, however, probably be greatly reduced by using the same efficient method for washing the drug from the nasal chamber soon after its application. In spite of the importance of the risk, it is difficult to see why these investigations should not be continued along the lines suggested, providing it is recognized and the rhinologist proceeds with caution. With a sensitive method for measuring the sense of smell (Elsberg method) the risk to volunteers willing to submit to preliminary tests may be further reduced by limiting the experimental treatments to one side of the nose only, so that if the worst should happen, the loss sustained would not be a total one.

We have been asked on several occasions what we knew about the loss and subsequent return of the sense of smell in monkeys following treatment with zinc sulfate solution. Such studies are difficult to carry out in monkeys, especially when dealt with in numbers sufficient to make the results significant and we have therefore not attempted such investigations. We can say, however, that the majority of treated animals eventually become again susceptible to intranasal inoculations of virus. This seems to offer indirect evidence that the olfactory receptors must eventually be restored. This tends to be borne out by our observations suggesting that the damaged olfactory epithelium gradually takes on the general appearance of normal olfactory epithelium. A full report on our histologic studies will appear later.

Before closing this discussion, I should like to say that we feel certain that investigations of other chemicals should be continued in the hope that a more suitable agent may be found. While zinc sulfate is highly effective, we are by no means certain that it is the most deserving agent for human trial. However, its effectiveness in animals has been more completely studied than that of any other substance and this fact in itself justifies its being kept in the foreground for the present.

Finally, I experience no little regret at being unable to write this article with a background of experience which would enable me to lay down in concrete terms the course to follow in the face of an epidemic of poliomyelitis. It is my hope, however, that in describing the problem as it stands and in presenting some of the difficulties associated with it, I have rendered a service to those of our colleagues who stand between the public and the experimental laboratory.

* Dr. Clarence A. Burgheim[17] of Managua, Nicaragua, has recently transmitted to me some information bearing on the application of zinc sulfate solution by the postural method. A case of poliomyelitis having appeared in Managua during the middle of June (1938), zinc sulfate prophylaxis was instituted immediately, a 1 per cent solution being applied by the Shahinian method.[13] The Nicaragua health service gave prophylactic treatments to 3,512 children and adults and Dr. Burgheim gave the treatment to 700 in his private practice. At least several hundred more treatments were given by other private physicians. Anosmia followed the treatments quite uniformly. It seems, however, that by August practically all had regained the function or were gradually recovering it. In Dr. Burgheim's own series, all between the ages of 6 and 15 years (187) had recovered the sense of smell by July 15 and only one in the adult group (127) remained anosmic, although an immediate anosmia had developed in every person treated. Functional tests were not possible on the group under five years of age (386). Since the Shahinian technique was followed in detail, these observations suggest that the incidence of permanent anosmias may not be any greater by the postural method than by the spray method.

With regard to the possible efficacy of zinc sulfate in controlling the disease little can be concluded from a single small epidemic. The impression gained, however, was that the prophylactic treatments had contributed materially to the rather abrupt ending of the epidemic. Twenty-six cases occurred in Managua within a period of fifteen days, all in children who had not received prophylactic treatments. The last case in Managua was reported on July 3. For Nicaragua proper, a total of thirty-seven cases was reported to the Director General of Health, the first case on March 31, the last on July 18. Two children given prophylactic treatments developed the disease; one seven days after treatment, and the other (brother of the first), eleven days after. Both of these therefore fall within the maximum incubation period of this disease. We hope that a full account of the observations in this outbreak will be published in due time.

REFERENCES

1. Schultz, E. W., and Gebhardt, L. P. Zinc sulphate as a chemoprophylactic agent in experimental poliomyelitis. *Proc. Soc. Exper. Biol. & Med.*, 35: 524, 1937; Olitsky, P. K., and Sabin, A. B. Comparative effectiveness of various chemical sprays in protecting monkeys against instilled poliomyelitis virus. *Ibid.*, 36: 532, 1937.
2. Schultz, E. W. Future of chemoprophylaxis as a measure for the practical control of poliomyelitis. *J. Pediat.*, 13: 38, 1938.
3. (a) Schultz, E. W. Correspondence. Bull. *Am. Acad. Ophth. & Otolaryng.*, 6: 9, 1937; (b) Schultz, E. W., and Gebhardt, L. P. The use of zinc sulphate solution for the prevention of poliomyelitis in man. Correspondence. *J. A. M. A.*, 110: 2024, 1938.
4. Schultz, E. W. and Gebhardt, L. P. Zinc sulphate prophylaxis in poliomyelitis. *J. A. M. A.*, 108: 2182, 1937.
5. Schaeffer, J. P. The Nose and Olfactory Organ. Phila., 1920. Blakiston. Special Cytology. Ed. by E. V. Cowdry. N. Y., Hoeber. 1: 47, 1928.
6. Sabin, A. B., and Olitsky, P. K. Mode of action of zinc sulfate spray in preventing infection with nasally instilled poliomyelitis virus. *Proc. Soc. Am. Bact., J. Bact.*, 35: 44, 1938.
7. Armstrong, C. Experience with the picric-acid-alum spray in the prevention of poliomyelitis in Alabama, 1936. *Am. J. Pub. Health*, 27: 103, 1937.
8. Peet, M. M., Echols, D. H., and Richter, H. J. The chemical prophylaxis for poliomyelitis. *J. A. M. A.*, 108: 2184, 1937.
9. Tisdall, F. F., Brown, A., DeFries, R. D., Ross, M. A., and Sellers, A. H. Zinc-sulphate nasal spray in the prophylaxis of poliomyelitis. *Canad. Pub. Health J.*, 28: 523, 1937.
10. Hayden, A. A. Zinc sulfate intranasal spray technic in poliomyelitis prevention. *M. Clin. North America*, Jan. 1938.
11. Echols, D. H., Richter, H. J., and Peet, M. M. The effect of zinc sulphate on the sense of smell. *Univ. Hosp. Bull., Univ. of Mich.*, 3: 32, 1937.
12. Pentecost, R. S. Zinc sulphate as a chemoprophylactic agent in epidemic poliomyelitis. *Canad. Pub. Health J.*, 28: 493, 1937.
13. Shahinian, L., Bacher, J. A., McNaught, R. C., and Newell, R. R. Chemoprophylaxis in poliomyelitis. The technic of applying chemical agents to the olfactory mucosa. *J. A. M. A.*, 110: 1254, 1938.
14. Furstenberg, A. C., and Hauser, I. J. Zinc sulphate. *Bull. Am. Acad. Ophth. & Otolaryng.*, 7: 9, 1938.
15. Elsberg, C. A., and Levy, I. Sense of smell: new and simple method of quantitative olfactometry. *Bull. Neurol. Inst., New York*, 4: 1, 1935.
16. Tisdall, F. F., Brown, A., and DeFries, R. D. Persistent anosmia following zinc sulfate nasal spraying. *J. Pediat.*, 13: 60, 1938.
17. Burgheim, C. A. Personal communication.

APPLIED PHYSIOLOGY OF THE NOSE AND THE ACCESSORY NASAL SINUSES*

ARTHUR PROETZ, M.D.

Professor of Clinical Otolaryngology, Washington University School of Medicine

ST. LOUIS, MISSOURI

TREATMENT of the nose and sinuses has always concerned itself with the eradication of infection and the establishment of ventilation and drainage.

If viewed from a purely mechanical standpoint, the simplest and most effective means of accomplishing these ends would be a complete removal of diseased tissue and the widest opening of constricted passages. In other words, the most radical operation which did not encroach upon the surrounding vital structures would best meet the requirements.

From a functional standpoint, however, such an attack would meet with failure because it would impair or destroy functions which are essential to the health and safety of the individual. The ideal treatment is one which will supply the required ventilation and drainage with the least possible damage to the physiologic processes of the nose.

Let us examine these processes.

First of all, the nose must warm the inspired air in order to protect the delicate linings of the pulmonary alveoli. It has been shown that in this respect it functions so effectively that the air has reached a temperature very near 37°C. before it enters the trachea. Helmholtz estimated that 70,000 calories of heat are radiated in the process.[1] It is known that, because of this radiation, the temperature of the nose remains about 30°C.[2] The heat is distributed through the extensive blood spaces of the turbinal bodies and it is thought that the sinus cavities may act as insulators against this cooling.

It is the function of the nose, further, to humidify the air. It does this equally effectively, supplying moisture to the point of saturation before the air reaches the trachea. This humidification is essential to the processes of alveolar respiration and nearly a liter of water is given off by the nasal mucosa in twenty-four hours.[3] This humidity is vital also to the maintainance of the nasal cilia and their protective mucous blanket, which constitutes what is probably the most important defense of the individual against infection. The ciliated epithelium plays a much more important part in nasal hygiene than was formerly supposed. Contrary to the earlier teachings, these structures are vigorous, difficult to destroy, and in health constitute an unbroken blanket, a moving conveyor from all parts of the nose and the accessory sinuses to the pharynx. In man, the cilia are not more than 7 micra in length but they move so vigorously and with such speed that the mucous blanket carrying foreign particles may be made to travel over them a distance many times their own length per second.

Definite pathways of ciliary streaming exist, the streams moving always from smaller to larger cavities, traveling always backward into the nasal chambers.[4,5] It is estimated that a particle is conveyed from the most remote recesses of the farthest sinus to the pharynx in thirty minutes.[6] Obviously incubation of bacteria in these areas is impossible so long as the blanket is unbroken.

One has but to study moving cilia under the microscope to be convinced of their extreme importance. In nature, they have but one enemy, namely drying. Wherever cilia exist, they must move in a liquid

* From the Department of Otolaryngology, Oscar Johnson Institute, Washington University School of Medicine, St. Louis.

medium. In aquatic animals this is to be found in the environment. In air-breathing animals, moisture must be richly supplied by the epithelial glands. Any deviation or obstruction in the nose of such a nature as to concentrate air currents in certain areas, thereby producing localized dry spots, results in destruction of cilia and impaired nasal health.[7]

Injudicious removal of nasal membranes may have the same result. This should not be misinterpreted to infer that no nasal tissues may be removed with benefit. However, a knowledge of respiratory air currents in the nose will materially assist one in planning such removals in such a way as to prevent drying and metaplasia.

The cilia are remarkably resistant also to the various medicaments usually employed in the nose. To this rule there are two notable exceptions. Cocaine solution stronger than 5 per cent and epinephrine solution stronger than 1:10,000 slow the ciliary beat immediately and stop it altogether in a very few minutes, usually beyond hope of resuscitation.[8,9] One must choose between the benefits to be derived from such solutions and the deleterious effect of a temporary cessation of ciliary action.

Mention has just been made of respiratory air currents in the nose. Unless some obstructive deformity or lesion occurs, these follow a fairly definite pathway. Inspired air passes from the nostril in a high curve through the olfactory fissure to the face of the sphenoid and down into the pharynx. Relatively little of it passes through the inferior meatus.[10] Expired air follows much the same curve. There is this difference, however, that in expiration the nostril, acting as a baffle, produces one large eddy and several smaller ones in the nose. Owing to the conformation of the anterior tip of the middle turbinate, the inspired air does not enter the middle meatus. The expiratory eddies, however, enter the meatus from behind.[11] It is significant that the ostia of the sinuses lying deep in the middle meatus are not subjected to dry, cold, dusty inspired air but only to warm, moistened air from the lungs.

Studies of air pressures in the nose and the sinuses have taught us that the air exchange between the two is extremely slow. Normal respiratory pressure fluctuations in the neighborhood of the ostia do not exceed 10 mm. of water pressure, or approximately $\frac{1}{1000}$ of an atmosphere.[12] If we discount diffusion, 1,000 respiratory cycles are required to effect a complete exchange of air in any sinus or, in point of time, approximately one hour.

Thus, it will be seen that since only the most negligible of air currents pass through the ostia, in and out of the sinuses, very little moisture is required to maintain the humidity within. This is reflected in the scarcity of glands normally to be found within the sinuses, whereas the nasal mucosa, which is exposed to air currents, is crowded with them. It may be significant also that the neighborhood of the ostium, where the air exchange takes place, is somewhat better supplied with glands than the remainder of the sinus.

In planning sinus surgery, it will be well to refrain as far as possible from creating openings which will permit a direct blast of inspired air to enter the sinus and dry the membrane. Such a procedure results in the immediate destruction of cilia, a metaplasia of the epithelium to something resembling the squamous type, with the result that the sinus is forever prevented from cleansing itself effectively. Such a sinus is a constant source of annoyance to the patient, through retained and frequently infected secretions, and irritative headaches. This condition is encountered chiefly after widely opening the face of the sphenoid, as such an opening is in the direct line of the inspired air current. A smaller opening, made near the septum will suffice for ventilation and irrigation and prevents this drying. It has also the advantage that it does not interfere with the ostium, which I believe should be regarded as a functioning organ and not

merely an opening. It is my experience that an undisturbed ostium can be returned to a much better functioning state than one embarrassed by scars following an attempt to enlarge it.[13]

It is granted, of course, that sinuses exist so crowded with irreparably infected mucosa that nothing is left but to remove it. This amounts to an amputation and it is not to be expected that normal function can again be reestablished. However, Wright[14] and others have shown that true hyperplasia is a long time in developing and that many thickened membranes can be made to return to approximate normality under proper treatment.

Operations in which sinus interiors are made accessible through openings in protected areas without disturbing the ostium are typified by the antrum "window" as it is commonly made under the inferior turbinate. Here there is no question of directing an inspired air stream into the cavity and the operation is usually effective.

Scar tissue is notoriously unmanageable in the nose, as anywhere else, and it is good practice to avoid it both by making the incision cleanly, with biting and cutting rather than crushing and tearing instruments, and by limiting such incisions to the extent required. A ragged opening, however large, has a greater tendency to close than a small one with a clean margin.

Medication in sinus disease often fails because it is badly chosen. Much of it in common use flies directly in the face of the physiologic demands of the nose. Some of it is ineffectual in that it fails to reach the diseased tissues. In this respect, the much maligned medicine dropper is the most effective means of application. Critics of the medicine dropper usually have axes to grind, but it is significant that agencies interested in the prevention of poliomyelitis report that fluids introduced into the nose with a dropper reach the cribriform plate when other methods fail. This has been repeatedly demonstrated by radiogram.

How shall nasal medication be planned in order to further the physiologic processes and not to hinder them?

As stated at the beginning, the chief aim of any nasal treatment is ventilation, drainage and the eradication of infection. The requirements for these three things are an unbroken ciliary blanket and a free airway through which it can function. Cilia are present and active even upon membranes bathed in pus. It requires remarkably little in the way of cleansing to induce them to regenerate upon areas in which they have been destroyed. If this can be supplied and at the same time the ostia can be kept patent a large step has been taken toward the recovery of the sinus. In his anxiety to do everything at once that can be done, one must guard against introducing substances such as the stronger antiseptics which are deleterious to ciliary activity. It is patently impossible to reach with antiseptics bacteria which are growing in the depths of the mucosa, or in the glands. If in attempting to do so one incapacitates the surface cleansing mechanisms, then one hinders recovery.

Fortunately ephedrine and its related compounds, in the customarily employed concentrations, have no slowing effect upon the cilia[8,9] and are at the same time active in opening the ostia. In our experience dilute solutions of this nature, introduced into the sinuses at frequent intervals (every other day) constitute the best single remedy in the long protracted subacute, and the chronic types of sinusitis. Displacement[15] is the method of preference since it is free from trauma—an important consideration when frequent treatments are necessary. Naturally this does not apply to the hyperplastic conditions in which the sinuses are crowded with polypoid growths. It does apply, however, to many cases which are generally considered unmanageable and are subjected to operative procedures.

As stated, most antiseptics have some slowing action on the cilia and produce more or less pain and irritation when

introduced into the sinuses. Their use is regarded favorably in some quarters, and I have no wish to condemn them, but in my own hands they have added nothing to what could be accomplished with the vasoconstrictors alone.

Oils as vehicles for nasal medication have no physiologic basis. On the contrary, they may be a source of danger to the lung. Custom alone seems to account for the continued use of oils, in the face of the facts that they are not miscible with the nasal secretions, which are 95 per cent water, that their affinity for the dissolved substances is sometimes so great that they do not release them to the tissues, and that the substances ordinarily found in oily mixtures have been shown to be irritants to the mucosa and nothing more.[16]

Reports of so-called lipoid pneumonia following the use of mineral oil drops and sprays in the nose have been appearing with increasing frequency. The retention of oil in the lung is particularly dangerous in infancy and old age.[17,18] Abundant experimental evidence is at hand to show that the condition is purely the result of the instilled mineral oil. Vegetable oils are ultimately absorbed, but so slowly that they may still be a source of danger.[18]

The past few years have witnessed a healthy revival of interest in the physiology of the nose. The resulting research has brought out many new facts which have thus far only succeeded in producing a fluidity of thought. More time will be required before it can be determined to what extent laboratory experiments upon animals may be applied to clinical problems in human beings. Mature consideration will possibly establish a better balance between harmful processes which are justified by the results which they accomplish and those which are not. In a series of articles such as this one, I trust that one is expected to draw upon his own experience rather than to summarize the accepted views of others (which after all is the function of textbooks) and this I have done. In conclusion I should like to observe that while it is easy and natural to apply new experimental evidence to our clinical practice, it is somehow a painful process to abandon familiar clinical procedures after they have been shown to rest upon false premises. Both are necessary to progress.

REFERENCES

1. Helmholtz. In Stewart. Manual of Physiology. 5th Ed., p. 502. Philadelphia, 1905. W. B. Saunders.
2. Cone, A. J. Variations in the temperature of the mucous membrane of the nose. *Arch. Otolaryng.*, 17: 66 (Jan.) 1933.
3. Aschenbrandt. In St. Clair Thomson. Diseases of the Nose and Throat. 4th Ed. New York, 1937. D. Appleton-Century Co.
4. Yates, A. Lowndes. Methods of estimating the activity of the ciliated epithelium within the sinuses. *J. Laryng. & Otol.*, 39: 554 (Oct.) 1924.
5. Lucas, A. M. Direction of ciliary movement in the nasal cavity of macacus rhesus. *Tr. Am. Laryng., Rhin. & Otol. Soc.*, 1931, p. 172.
6. Proetz, A. W. Principles of sinus treatment in the light of physiologic facts. *Tr. Sect. Laryng., Otol. and Rhin., A. M. A.*, 1933, p. 3.
7. Proetz, A. W. Nasal ciliated epithelium, with special reference to infection and treatment. *J. Laryng. & Otol.*, vol. 49, Sept. 1934.
8. Proetz, A. W. The effects of certain drugs upon living nasal ciliated epithelium. *Ann. Otol., Rhin. & Laryng.*, 43: 450 (June) 1934.
9. Lierle, D. M., and Moore, P. M. Effects of drugs on ciliary activity of mucosa of upper respiratory tract. *Arch. Otolaryng.*, 19: 55 (Jan.) 1934.
10. Lack, Lambert. In St. Clair Thomson. Diseases of the Nose and Throat. 4th Ed. New York, 1937. D. Appleton-Century Co.
11. Proetz, A. W. Some intimate studies of nasal function: their bearing on diagnosis and treatment. *Ann. Otol., Rhin. & Laryng.*, 41: 125 (March) 1932.
12. Braune and Clasen. Die Nebenhöhlen der Menschlicher Nase, etc. *Ztschr. f. Anat.*, 2: 1, 1877.
13. Proetz, A. W. Treatment of sinus disease. *Pennsylvania M. J.*, March 1936.
14. Wright, Jonathan, and Smith, Harmon. Diseases of the Nose and Throat. Philadelphia, 1914. Lea and Febiger, p. 109.
15. Proetz, A. W. The Displacement Method of Sinus Diagnosis and Treatment. St. Louis, 1931. Annals Publishing Co.
16. Fox, Noah. Effect of camphor, eucalyptol and menthol on the vascular state of the mucous membrane. *Arch. Otolaryng.*, 6: 112 (Aug.) 1927.
17. Laughlen, G. F. Studies on pneumonia following nasopharyngeal injections of oil. *Am. J. Path.*, 1: 407, 1925.
18. Walsh, T. E., and Cannon, Paul R. The problem of intranasal medication. *Ann. Otol., Rhin. Laryng.*, 47: 3 (Sept.) 1938.

PATHWAYS OF REFERRED PAIN FROM THE NOSE*

RALPH A. FENTON, M.D.

Clinical Professor of Otolaryngology, University of Oregon Medical School

PORTLAND, OREGON

REFERRED pain has come to include not only stimuli which travel upward by the ordinary cerebrospinal nerves of conscious sensation, but also discomforts due to irregularities of vascular, glandular and muscular tonus, perceived as pain by sensory nerves, but referred thereto by the autonomic nervous system. Thus the injunction of Hilton, in his fourth lecture on "Rest and Pain," to trace the nerves of and from the particular painful spot, mentioning the fifth cranial nerve as the true cranial sensation nerve, has acquired a complexity unforeseen in 1860.[1]

Considering first the direct route, it is of course apparent that relative simplicity of the distribution of the mixed cranial nerves in fishes has in higher species and the human become vastly more complex owing to two factors: first, the loss of either sensory or motor components from these early mixed nerves, so that in the human species the fifth is very largely sensory; and second, enormous development forward of the cerebral hemispheres, pressing the face down from above, while the orbits have been turned forward to make binocular single vision possible. This approach of the orbits to each other reduces the size of the nasal airway, accessory sinuses and olfactory area. Such narrowing makes it easy for painful pressure to occur.

External deformities or fractures, and internal obstruction such as crowding of the turbinates against septal deviations or thickening may produce more or less serious local pressure, and also interfere with normal sinus drainage. Since these areas are all supplied by the fifth nerve (Fig. 1), pressure pains may be referred through the Gasserian ganglion as follows: (1) from the external nose via the infratrochlear and external nasal branches of the nasociliary nerve, which comes from the ophthalmic division; and (2) from the mucous membranes of the accessory sinuses, septum and turbinates, through the maxillary division, traversing the sphenopalatine ganglion and forming the palatine and sphenopalatine nerves.

Direct reference of pain by these pathways depends on the presence of twigs from the ophthalmic division to the tentorium cerebelli, from the maxillary division to the dura of the anterior fossa of the skull, as well as upon irritation of the cutaneous distribution of the fifth nerve. For example, frontal sinusitis gives supraorbital pain; maxillary sinusitis, if acute, may give pain over the cheek, but more commonly and as it becomes more chronic, gives supraorbital headache; anterior ethmoiditis causes pain at the inner canthus and in the parietal region up to the vertex; while the posterior ethmoid and sphenoid may arouse parietal or vertex pain but are more likely to produce otalgia, not only in the external auditory meatus but also in the mastoid and occipital region.

This last syndrome, known as sphenopalatine or Vidian neuralgia, is the most complex of all the manifestations of nasal irritation. Its clinical implications include not only diseases of the posterior group of nasal sinuses and high thickenings of the septum, but also allergic swelling of the mucosa. Remote influences may lead to turgescence of the middle turbinate, particularly in women preceding the menstrual period or at the climacteric; such disturbances may point to uterine displacements or fibroids, pelvic adhesions, or chronic colonic stasis. The effects of latent muscu-

* From the Department of Otolaryngology, University of Oregon Medical School, Portland, Oregon.

lar imbalance or refractive error may be translated into stimuli passing through this area of the nose.

pinna of the ear, the skin over the mastoid, and a variable area within the pinna and along the posterior wall of the external

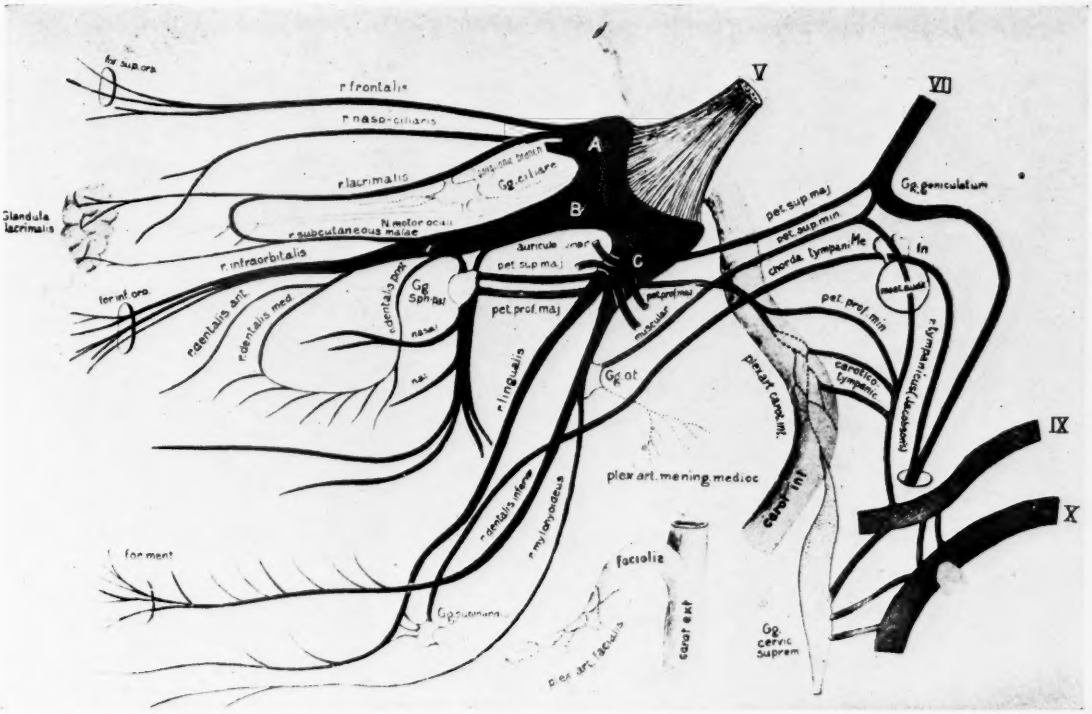


Fig. 1. Interrelation of the fifth, seventh, ninth and tenth cranial nerves, together with the sympathetic ganglia in man. (From Wilder, H. H. History of the Human Body. Henry Holt.)

Sluder[2] long ago exhaustively studied this area from the clinical standpoint without advancing adequate anatomic reasons for reference of pain to the ear, occiput and shoulder. The explanation was foreshadowed thirty years ago by Ramsay Hunt's[3] pioneer work on the neurology of herpes zoster affecting the ear; but it remained for Larsell[4] to offer microscopic and anatomic proof of the route followed by these painful stimuli.

The sphenopalatine ganglion (Fig. 2) is connected not only with the maxillary division of the fifth, but has a large posterior branch, the Vidian, which divides into the great deep and great superficial petrosal nerves. The latter contains large myelinated fibers of sensory type, which connect in the geniculate ganglion with the sensory distribution of the facial nerve.

This distribution corresponds exactly with the areas involved in herpes zoster oticus, namely the posterior surface of the auditory meatus.[3] A direct sensory nerve connection is thus seen to exist between the sphenopalatine region of the nose and the skin of the ear. Clinical discrimination between these sharp pains and actual ear or mastoid involvement offers little difficulty, since cocainization of the ganglion area ordinarily arrests this symptom.

No such direct pathway exists, however, for referred pain to the trapezius muscle, occipital and deltoid regions. Hence, Larsell followed his research of 1928 with further studies directed toward the vasomotor innervation of the nose.[5]

Just as pain may occur in an extremity owing to interference with its vascular supply, being referred to the appropriate spinal segment even though the disturbance arises through the autonomic system, it was reasoned that similar vascular difficulties in the very complex head and neck region might be projected into the sensory domain of some cervical nerves.

Vasodilator and secretory fibers reach the nasal mucosa from the Vidian nerve through the sphenopalatine ganglion. Vaso-

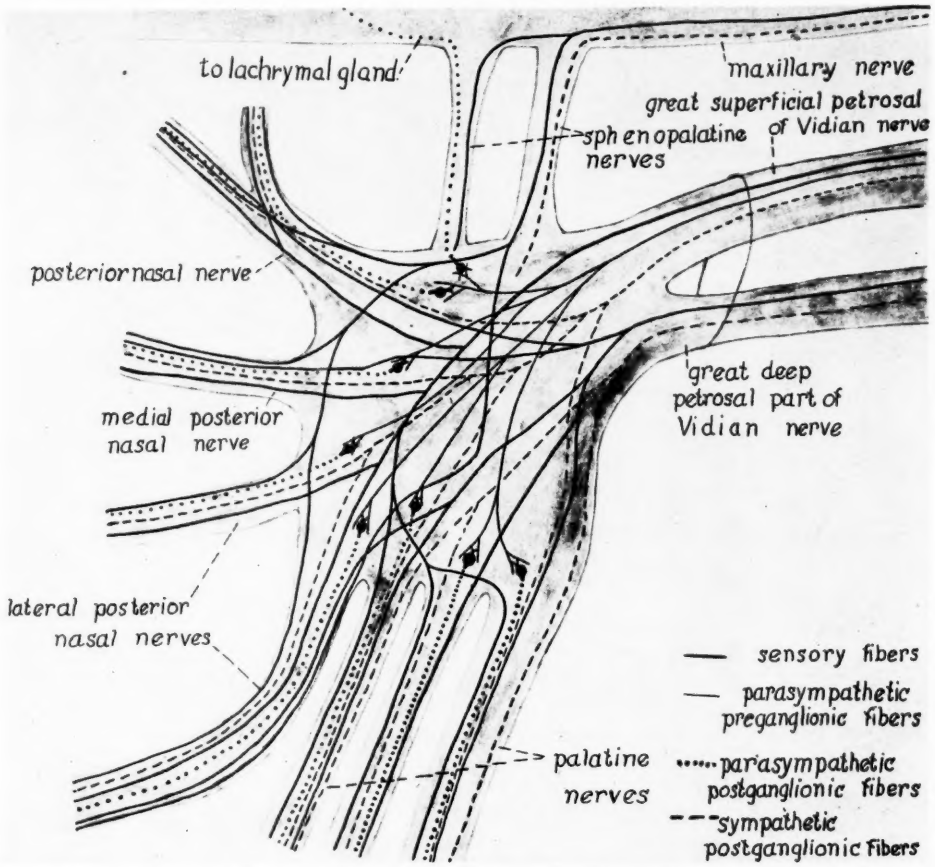


Fig. 2. The sphenopalatine ganglion and its connecting fibers. (From Larsell and Fenton, in *Arch. Otolaryng.*, 24: 687, 1936.)

constrictor impulses seem to travel upward from the superior cervical sympathetic ganglion to and through the sphenopalatine, while vasodilatation and secretion seems largely to be controlled by fibers which form a part of the great superficial petrosal nerve.[6] (Fig. 3.)

Experimental evidence based on local anesthesia and ischemia of the mastoid skin in persons suffering from referred pain from the sphenopalatine region—anesthesia blocking such referred pain and ischemia producing it—suggested further examination on the circulation of the rabbit's ear.

It was found, after section of the maxillary nerve, thus excluding the fifth, that faradic stimulation of the sphenopalatine region produced marked vasoconstriction in the ear.[7] These impulses must have traveled by the posterior nasal, great superficial petrosal, and possibly the deep petrosal nerve. Vasoconstriction was abolished, however, by section of the cervical part of the spinal cord at the level of the sixth spinal segment, or by section of the cervical sympathetic trunk between the middle and inferior cervical sympathetic ganglia.

Since these regions of the neck and shoulder are affected by reflex ischemia actuated by stimulation of the sphenopalatine area of the nose, pain is referred to the ischemic areas, as elsewhere in the body. The nervous pathways involved are therefore the nasal and palatine branches of the sphenopalatine ganglion, the great superficial petrosal nerve, the connections within the medulla oblongata with the reticulospinal tract (the reticulospinal path

arises from generalized cells in the medulla oblongata having to do with the conduction of vasomotor and other sympathetic tract make connections with the preganglionic fibers of the sympathetic vasoconstrictors which arise from the lower part

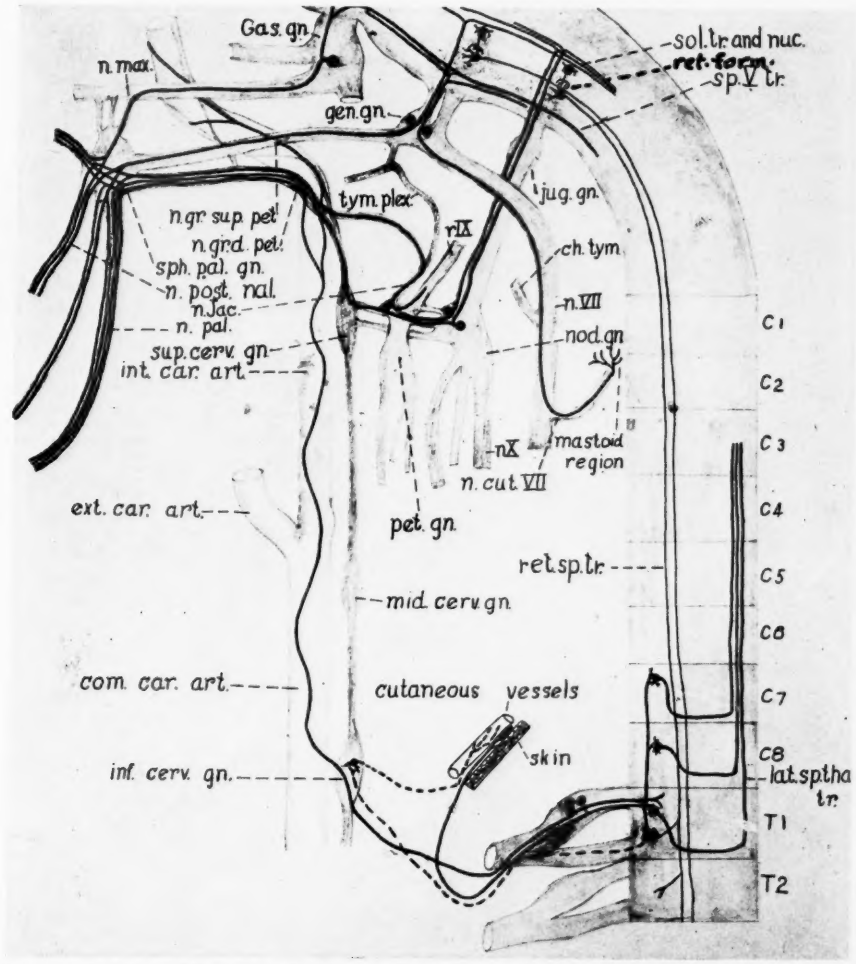


FIG. 3. Afferent fibers in relation to the sphenopalatine ganglion. The cutaneous vasomotor reflex from the upper thoracic segments and the relations of segmental afferent fibers are shown, indicating their central connections. Abbreviations: *cb.tym.*, chorda tympani; *com.car.art.*, common carotid artery; *ext.* and *int.car.art.*, external and internal carotid arteries; *gas.gn.*, gasserian ganglion; *gen.gn.*, geniculate ganglion; *inf.mid.* and *sup.cerv.gn.*, inferior, middle, and superior cervical sympathetic ganglions; *jug.gn.*, jugular ganglion; *lat.sp.thal. tract*, lateral spinothalmic tract; *n.cut.VII*, cutaneous branch of the facial nerve; *n.max.*, maxillary branch of the trigeminal nerve; *n.gr.sup.pet.*, great superficial petrosal nerve; *n.gr.d.pet.*, great deep petrosal nerve; *n.Jac.*, nerve of Jacobson; *nod.gn.*, nodose ganglion; *n.pal.*, palatine nerve; *n.post.nas.*, posterior nasal nerves; *n.VII*, facial nerve; *n.IX*, glossopharyngeal nerve; *n.X*, vagus nerve; *pet.gn.*, petrosal ganglion; *ret.form.*, formatic reticularis; *ret.sp.tr.*, reticulospinal tract; *r.IX*, root of glossopharyngeal nerve; *sol.tr.* and *nue.*, tractus and nucleus solitarius; *spb.pal.gn.*, sphenopalatine ganglion; *sp.V.tr.*, tract of fifth spinal nerve; *tym.plex.*, tympanic plexus. (From Larsell and Fenton, in *Arch. Otolaryng.*, 24: 687, 1936.)

stimuli) and, through those, the connections with the segmental autonomic nerves in the lower cervical segments of the cord. Impulses passing along the reticulospinal of the cervical and the upper part of the thoracic portion of the spinal cord, thus affecting the arteries of the ear and adjacent structures.

It is therefore unnecessary to ascribe to the cervical sympathetic any rôle in pain transmission save its well-established function of vasomotor and secretory regulation. But when, in the nose, interference with normal autonomic regulation occurs, the altered situation may be referred to distant regions in the ear, neck and shoulder through the cervical sympathetic and its anterior-root connections with the spinal cord and up to the brain.

Tracing of these pathways for pain from the nose requires accurate history-taking as well as careful local examination.

One should not, in these cases resort on insufficient evidence to radical surgery; and one may well remember that S. Weir Mitchell[8] cautioned us, more than sixty years ago, to beware of the easy alternative in diagnosis—hysteria or neurasthenia.

REFERENCES

1. Hilton, John. On Rest and Pain. 2nd ed., New York, 1879, p. 41.
2. Sluder, G. Nasal Neurology, Headaches, and Eye Disorders. (bibl.) St. Louis, 1927, p. 100.
3. Hunt, J. Ramsay. Herpetic inflammations of the geniculate ganglion; a new syndrome and its complications. *J. Nerv. & Ment. Dis.*, 34: 73, 1907.
4. Fenton, R. A., and Larsell, O. Embryology and neurohistology of sphenopalatine ganglion connections. (Casselberry Prize Research, Am. Laryng. Assn.) (bibl.) *Ann. Otol., Rhin. & Laryng.*, 42: 739, 1928.
5. Larsell, O., and Fenton, R. A. Sympathetic innervation of the nose. (Research for the American Academy of Ophth. and Otolarng) (bibl.) *Arch. Otolaryng.*, 24: 687, 1936.
6. Blier, X. Physiology of the sphenopalatine ganglion. *Am. J. Physiol.*, 98: 398, 1930.
7. Larsell, O., Barnes, M. S., and Fenton, R. A. Relation of irritation in region of paranasal sinuses to certain vasomotor changes. (Research for the American Academy of Ophth. and Otolaryng.) (bibl.) *Arch. Otolaryng*, 27: 266, 1938.
8. Mitchell, S. Weir. Notes on headaches. *Phila. Med. & Surg. Rep.*, 32: 101, 1875.

During the early part of the Renaissance, postmortem examinations were performed before anatomic dissection had been instituted.
From—"The Postmortem Examination" by Sidney Farber (Thomas).

EXTERNAL OPERATIONS ON THE FRONTAL SINUS

HAROLD I. LILLIE, M.D.

Section on Otolaryngology and Rhinology, The Mayo Clinic

ROCHESTER, MINNESOTA

MANY articles describing the technical procedures suitable for external operation on the frontal sinus have appeared in medical literature. In some instances, emphasis has been placed on technical details rather than on the indications for and the choice of surgical procedures that would be adequate for the pathologic condition encountered. In operations on cavities that have rigid walls, the general principles laid down long ago by Küster still obtain.

In this article I propose to express my personal opinions concerning the reasons for choosing a certain type of external operative procedure for certain pathologic conditions of the frontal sinus. Because details of surgical procedures are, and should be, personal, and as technical adaptations of the surgeon should conform to the underlying general principles of adequate surgical attack, I do not propose to emphasize technical details except as they may seem to be important under specific circumstances. There seems to be no point in distinguishing between conservative and radical surgical interference, for in one instance a very formidable surgical attack may be necessary and actually be conservative for the disease encountered, whereas such an attack might be entirely unnecessary and misdirected under other circumstances. In other words, the surgical procedure must be adapted to the pathologic condition encountered; the pathologic condition must not be adapted to a surgical procedure.

ANATOMY

There are certain fundamental anatomic and pathologic factors that must be taken into consideration when the advisability of the use of an external operation on the frontal sinus is under consideration as a curative measure. It does not seem necessary to describe the anatomy of the frontal sinus. In the first place, it is impossible to do this accurately, and in the second place, the frontral sinus normally varies greatly in size, conformation, and position. The frontal sinuses develop, after birth, from projections of the ethmoidal cells into the frontal bone. This development may or may not be symmetrical. One frontal sinus may develop and the opposite one may be represented merely by a small projection of an ethmoid cell. On the other hand, the ethmoid cells may extend well under the frontal sinus to the lateral aspect of the orbit and as far posteriorly as the cone of the orbit. Reference to the diagrammatic sketches (Fig. 1) will reveal the various types of frontal sinuses that may be encountered. Externally, there may be no indication of the type of frontal sinus with which one is confronted except perhaps that if the frontal bosses are prominent, it can be inferred that the anteroposterior diameter of the sinus is large.

Transillumination has been used as an aid in the diagnosis of conditions of the frontal sinus, but the method is not sufficiently accurate to be very useful. The Roentgen rays furnish the best information so far as the anatomic features of the frontal sinus are concerned. It is important that this information be had before contemplating any operation on the frontal sinus because one will then be forewarned as to the presence of divisions in the frontal sinus and to the extent of the operation necessary to expose adequately the interior of the sinus. Interpretation of the roentgenograms should be very exacting. One must determine the thick-

ness of the outer table and the inner table, the presence or absence of fronto-ethmoid cells and their extent, divisions within the sinus, or the absence of a frontal sinus. In addition to this information, it is important to know the relative measurement of the space between the nasal septum and the inner canthus of the orbit because when this space is wide, fashioning of a large nasofrontal duct is relatively more easy to accomplish than it is when this space is narrow. (Fig. 1.) The injection of opaque medium into the frontal sinus before roentgenologic examination is very helpful if the medium can be distributed throughout the interior of the sinus.

The diagnostic judgment of the surgeon remains the important standard in determining the operative attack that will be adequate for the anatomic conditions encountered. For instance, it might be impossible to expose adequately the interior of a frontal sinus of the extensive multilocular type in which the anteroposterior diameter is very small, as shown in Figure 1 (6 and 9). The Killian type of operation would hardly be necessary in a small symmetrical type of frontal sinus, as shown in Figure 1 (4).

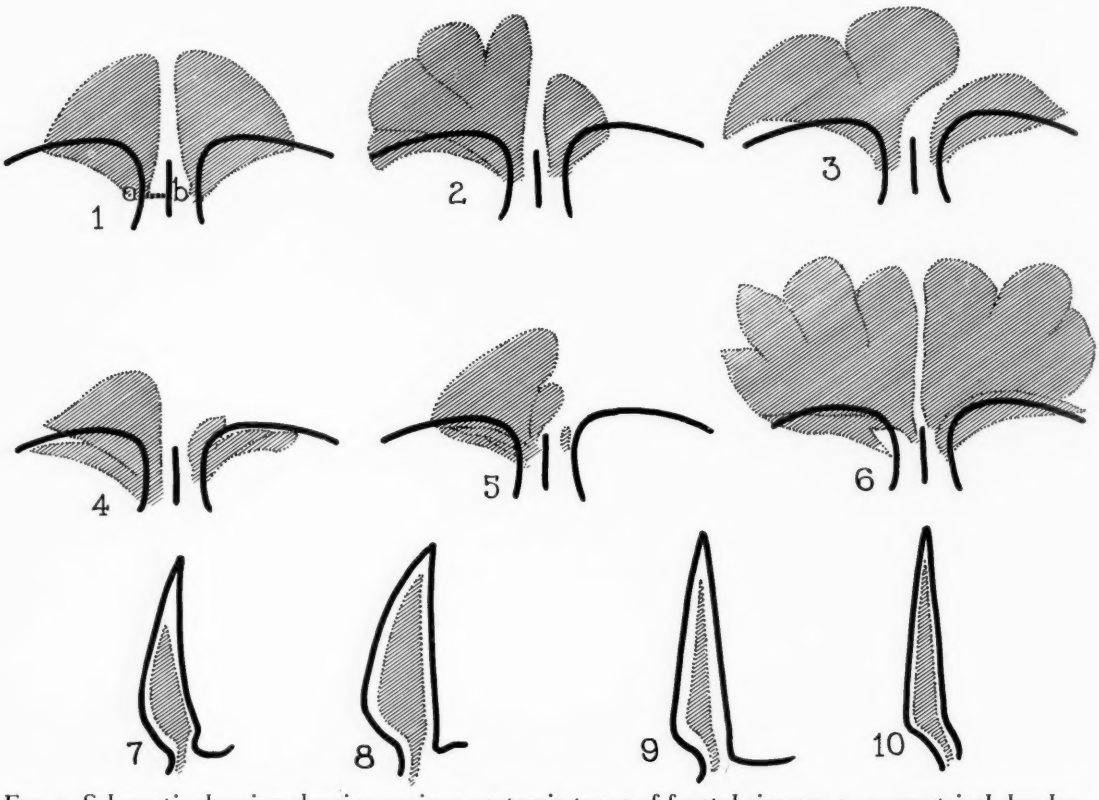


Fig. 1. Schematic drawing showing various anatomic types of frontal sinuses: 1, symmetrical development of sinuses (anterior view); line *a-b* indicates distance between nasal septum and inner canthus of the orbit; 2, 3 and 4, asymmetrical development of sinuses (anterior view); 5, absence of one frontal sinus (anterior view); 6, large multilocular type of frontal sinus (anterior view); 7 and 8, type of sinus in which Hansen type of operation would afford adequate exposure from below (anterior and lateral views); 9 and 10, type of sinus which would require Killian type of operation for adequate exposure, particularly if the sinus were multilocular (lateral views).

It would seem that absence of a frontal sinus might be easy to determine in the roentgenogram. However, Skillern recently has shown that, as the result of an obliterating osteitis, the bone undergoes a reparative process and that marked sclerosis, similar to that which occurs in the mastoid bone in the presence of chronic infection, may occur. In reality, there may be cavitation within this sclerosing process which may not be discernible in

the anterior or lateral roentgenogram. In such an instance the diagnostic ability of the surgeon is severely taxed.

PATHOLOGIC CONSIDERATIONS

Compared to the mucous membrane lining the nasal cavity, the mucous membrane lining the interior of the paranasal sinuses is different in several respects. First, it is very much thinner. The submucosal layer is practically lacking, the extensive vascular spaces and erectile tissue are absent, and excepting near the nasofrontal duct, there are few secreting glands. The periosteum and fibrous layer are difficult to distinguish from each other.

When the mucous membrane is involved by an inflammatory process, intense swelling occurs. The mucous film covering the ciliated epithelial cells is increased, the epithelial cells undergo marked cloudy swelling, the underlying membrane is edematous and infiltrated with leucocytes, and the small blood vessels are greatly increased in size. The swelling of the membrane may be so great that the cavity of the sinus is filled. Intense pain results in small sinuses, because of the pressure due to the swelling of the membranes and the accumulation of secretion. If the nasofrontal duct is small and its opening constricted to the extent that the cilia cannot move the accumulated secretion out of the sinus, pain is greatly increased.

If such a state exists for some time, necrosis of the epithelial cells takes place; this extends to the underlying membrane and then to the periosteum. If the infection is particularly severe, thrombophlebitis extends into the bone and causes osteomyelitis. If the natural reparative process gets the upper hand and if the extension of the infection is limited, localized necrosis of the bone takes place. This is followed by involvement of the periosteum on the outer surface of the frontal bone. External manifestations include redness and swelling and, later, the formation of a subperiosteal abscess. When the infection is not extremely virulent, that is, should necrosis and rapid extension of the infection not occur, other definite physical changes take place in the lining membrane. Localized or generalized hyperplasia and hypertrophy of the constituent elements are common pathologic changes. In addition, if the glandular arrangement is especially extensive, cystic degeneration of the membrane may take place. This finally results in polyposis or the formation of a mucocele or pyocele. The last two pathologic conditions may increase insidiously in size even after acute symptoms of sinusitis have disappeared. By their slow growth they may cause pressure necrosis of the walls of the sinus and produce external manifestations. This extension usually occurs toward the orbit, because the bone forming the floor of the sinus is thinner than the bone of the forehead. It may occur over the forehead, however. Occasionally mucoceles or pyoceles of enormous size are encountered.

Of the neoplasms that may be present, osteomas are the most common. Meningiomas may be confused with osteomas. The principal differential characteristic is that in the roentgenogram a meningioma does not appear as dense as does an osteoma. Osteoma and meningioma may be found accidentally in cases in which routine roentgenograms are made. It is not necessary to consider surgical removal of an osteoma of the frontal sinus unless definite symptoms and signs seem to demand such treatment.

Malignant lesions are rare as compared to the incidence of malignant lesions in the maxillary sinus.

That it is important to recognize the nature of the pathologic lesion is obvious, for on its recognition rests the choice of the surgical approach. In general, it may be said that if the pathologic lesion has not caused serious disease of the lining membrane or bone any surgical approach that will permit adequate drainage and ventilation of the sinus is adequate. If there is serious disease of the lining membrane and

the bone, the diseased tissue must be removed.

ANESTHESIA

Improvement in methods of anesthesia suitable for this type of operation has been a great advantage to the patient and surgeon. In many cases local anesthesia is very satisfactory because it tends to decrease the bleeding at the time of operation. It has not been found satisfactory for secondary operations because the presence of scar tissue prevents adequate anesthesia by infiltration. Occasionally, if infiltration proves inadequate, the local anesthesia can be supplemented with intravenous anesthesia to good advantage. Rectal anesthesia has many advantages over inhalational anesthesia produced by the open method, because of the tranquility of the patient and the even degree of anesthesia that it affords. In addition, the field of operation is not obstructed with anesthetic implements. Intratracheal anesthesia offers many advantages. The nasopharynx can be packed off, the intratracheal tube does not obstruct the operative field, the patient may be kept under light anesthesia, the amount of anesthesia can be accurately controlled, and following operation the period of recovery is much shorter than it is when rectal anesthesia is used.

EXTERNAL OPERATIONS

At the present time two types of external operation are employed in the treatment of chronic disease of the frontal sinus. The choice of operation depends on the anatomic configuration of the frontal sinus and on the nature of the pathologic process. If there is no disease of the external table and if the anatomic configuration of the frontal sinus is such that the interior of the sinus can be visualized, adequate operation can be performed by approaching the interior of the sinus through the floor. If there is definite evidence of involvement of the external table of the frontal sinus, approach to its interior through the floor is inadequate because it does not remove the diseased portion of the external table. In the past, fear of unsightly postoperative deformity has caused surgeons and patients alike to dread operations on the frontal sinus, and in some instances it no doubt has caused surgeons to withhold the surgical procedure that would have controlled the disease and avoided the danger of serious complication.

At the present time the method that originally was suggested by Jansen, later described by Lynch, and more recently elaborated by Sewall, Smith, and others, avoids the deformity in those cases in which there is no disease of the external table of the frontal sinus. Lynch emphasized the necessity for the complete removal of the bony floor of the frontal sinus and the fronto-ethmoid cells. Sewall and Smith strongly emphasized the importance of removing every vestige of mucous membrane within the cavity of the frontal sinus and removal of the fronto-ethmoid cells.

If disease of the external table of the frontal sinus is present, the Killian type of operation should be employed. The obviousness of the deformity that may result because the soft tissues drop abruptly into the obliterated frontal sinus may be avoided in a large measure by beveling the edges of the bone so that the soft parts will not fall abruptly into the depression. This suggestion which Anderson and I made in 1927 may have been made before, although I have not encountered the description in medical literature. The aim of the Killian type of operation is to produce obliteration of the cavity by scar tissue and, therefore, prevent recurrence of attacks of frontal sinusitis. This operation is successful in many cases. Under certain social conditions, for instance, when the patient does not live close to a medical center and it is necessary that he be rid of the disease in order to carry on his occupation, it would be advisable to consider the Killian type of operation, in spite of the possibility that it may result in some deformity, rather than to use an operation that would not cause obliteration of the frontal sinus. As a mat-

ter of fact, however, obliteration of the frontal sinus does occur in many cases in which the Jansen type of operation is used, particularly if the floor of the frontal sinus and if the fronto-ethmoid cells are so completely removed as to avoid the formation of pockets lined with mucous membrane. I have observed definite evidence of this obliterative process in two cases in which subsequent operation was performed because the floor at the angle of the orbit had not been obliterated completely by the first operation. At the secondary operation I found that the cause of the failure was a pocket which was lined by mucous membrane; this had been kept open by three bony walls of the outer portion of the sinus. Correction of this defect resulted in complete resolution. The remainder of the sinus was filled with fibrous scar tissue.

Occasionally, following the Jansen type of operation the sinus is not obliterated but remains a cavity, because epithelial cells extend into it from the nose and line the cavity completely before fibrous tissue forms. The process of epithelization is no doubt aided by the fact that small patches of epithelium have been overlooked at the time of operation. There can be no disadvantage if the nasofrontal duct remains large and patent so that the interior of the sinus is readily accessible to direct treatment.

Permitting the frontal process of the superior maxilla[5] to remain in place at the time of operation in a large measure helps to keep the nasofrontal duct open by preventing the soft parts from contracting into the duct, and thus tends to be a factor in causing the cavity of the frontal sinus to remain patent.

In order to explain satisfactorily why obliteration occurs in one instance and the frontal duct remains open in another, one must fall back on the hackneyed idea of individual tendency to repair. It is common experience in everyday surgical practice.

If it is deemed necessary that interference be instituted for the relief of the symptoms and signs in acute suppurative frontal sinusitis, the question arises as to whether or not the sinus should be emptied from within the nose or externally. To me, an external opening, as a trephine opening in the floor of the frontal sinus, seems advisable. When an external opening is made the danger of complication is much less than it is when an opening is made through the vascular region around the nasofrontal duct.

In cases of fulminating acute frontal sinusitis in which there is evidence of extension to the bone and spreading osteomyelitis of the frontal bone, the consensus now is that very radical interference should be instituted. The optimal time for instituting such a radical surgical measure is difficult to determine. The observations of Furstenberg, and Mosher and Judd demonstrate that the infection follows the route of the blood vessels.

In the subacute stage of a frontal sinusitis relief of symptoms in many instances can be afforded by adequate enlargement of the nasofrontal duct. If this does not provide amelioration, an external type of operation is necessary; the choice of operation depends on factors outlined in the discussion on the anatomic variations and pathologic changes within the frontal sinus. If, in addition to the pathologic changes within the sinus, there is evidence of external extension, either toward the orbit or over the forehead, it means that necrosis of the periosteum has extended through the bone and has produced necrosis of the bone, localized osteomyelitis or a subperiosteal abscess. In a case in which such a pathologic process is present, an external operation is indicated because ventilation and drainage are not adequate to control the disease process. The mucous membrane and all diseased bone must be removed and all bony pockets or divisions of the frontal sinus and fronto-ethmoid cells must be eliminated. In certain cases in which direct visualization through an ordinary opening in the floor of the frontal sinus, such as that afforded by the Jansen type of operation, is difficult, it is possible

at times to expose the interior of the sinus adequately by carrying the removal of the bone higher on the brow. With suitable bone-biting forceps and curettes, removal of membrane and periosteum from the bone may be successfully carried out. Smith advised the use of picric acid to remove every vestige of mucous membrane. In my experience, however, if the periosteum is seriously diseased, it may be separated rather easily from the bone. It is more important that the mucous membrane be removed in the large multilocular type of frontal sinus than it is in the symmetrical small type of frontal sinus. In this way obliteration of the cavity is more likely to take place and the formation of pockets will be avoided. If mucous membrane is left in any space of the frontal sinus, my experience has been that the danger of pocketing and continuation of secretion from this mucous membrane is eminent and that it will defeat an otherwise well directed surgical procedure. If, however, the limits of the frontal sinus cannot be adequately reached by the Jansen technique, the Killian type of operation may immediately be employed.

At times, it is difficult to expose the field by an incision in the brow and it may be necessary to make a vertical incision, which starts at the bregma and continues upward toward the hair line. This should be done because visualization of the interior is extremely important. The edges of the depression may be beveled with a suitable gouge or chisel and it is surprising how little deformity results when this is done. If both frontal sinuses are involved, exposure can be obtained by making the ordinary incision for each side, joining the two at the root of the nose and elevating the flap upward. The need for this type of exposure is imperative when there is evidence of external manifestation of disease over the forehead. The same obtains for localized osteomyelitis. Pastore and Williams recently have suggested an incision beginning at the temporal fossa on one side and extending upward over the forehead and downward on the opposite side to the opposite temporal fossa. Elevation of the flap downward over the forehead is then carried out. This affords a very adequate exposure in those cases in which spreading osteomyelitis is present.

When mucocele or pyocele of the frontal sinuses is present, the same general principles obtain as have been outlined. The pathologic process is essentially different and the tendency for resolution following adequate operation is great. The pathologic process in itself has had a very insidious effect on the involved structures. The external manifestation has been produced by pressure necrosis without tendency for spread of the infection. All that is necessary is that the mucocele or pyocele sac be removed and that an adequate opening through the nose into the frontal sinus be maintained.

When neoplasms of the frontal sinus are present, depending on their nature, operative interference suitable for the lesion suggests itself. Osteoma is the most frequently encountered neoplasm of the frontal sinus. Occasionally, no subjective symptoms have been present, there have been no external signs of extension and the neoplasm is discovered during a roentgenologic examination. Whether or not an osteoma that has been discovered in the course of roentgenologic examination should be removed unless it has been checked over a period of years for increase in size is a question. However, if the osteoma has grown so that there is external evidence of its presence and if symptoms are severe enough to warrant its removal, this can be accomplished readily by any operation that adequately exposes the field. Osteomas have arisen in the region of the ethmoid sinus and have entered every individual ethmoid cell; their extension may be compared to the progress that an ameba makes by a pseudopodium. Meningiomas have been encountered. They greatly resemble osteomas except that reoentgenologic examination discloses that they are not so dense; they may be sym-

metrical and as smooth as osteomas. I have observed two cases of carcinoma of the frontal sinus. In both cases treatment consisted of exposure of the lesion and the use of radium therapy.

POSTOPERATIVE CARE

The postoperative care of the wound is very important. If the operation has been meticulously and carefully performed, the external wound can be closed and drainage into the nose will be sufficient to take care of any accumulation of secretion. There is one exception to this situation. If the fronto-ethmoid cells extend well out toward the external canthus and over the curve of the orbital contents, a drain in the external angle of the wound facilitates proper healing. One may use any type of pack or drain in the frontal sinus. Occasionally, it is not necessary to pack the frontal sinus because there has been little bleeding. When general anesthesia has been used, it usually is necessary that the bleeding be controlled. One may expect that there will be considerable swelling of the soft parts following operation, more in certain cases than in others. This will occur particularly in those cases in which severe traction of the soft parts has been necessary to expose the operative field. Some of the swelling can be partially eliminated by releasing the traction occasionally. Formerly it was the custom to use hot compresses over the wound for the relief of the postoperative swelling. This method caused softening of the wound. The use of infra-red light serves the same purpose better because it allows the wound to be dry and prevents maceration of the skin and soft parts. The external bandage can be removed in two or three days. The sutures can be removed at the same time.

Because the removal of the pack that has been used to fill the cavity of the frontal sinus will cause considerable pain, intravenous anesthesia or adequate sedation should be used. Ordinarily, the cavity does not need to be repacked. Occasionally, because of the size of the cavity of the frontal sinus, it is necessary to reopen the wound at the time of the first major dressing in order to view the interior of the wound and remove the packing. A large rubber tube, preferably a large catheter with the bulb end in the frontal sinus, may be placed in the nasofrontal duct. It need not extend out of the nostril. Allowing this tube to remain in position for a considerable time insures the patency of the nasofrontal duct better than any other method.

In the postoperative care of this type of wound some surgeons prefer to use irrigations to remove any accumulation of secretion and discharge that may occur in the cavity. I much prefer the dry method of treatment which consists of removing the collected secretions by use of a suction cannula. I am certain that the postoperative course is less protracted under these circumstances than it is when irrigations are used. Occasionally, even with the dry method of treatment, it is important that the cavity of the frontal sinus be flushed out occasionally to remove residual blood clots and débris. The recurrence of granulations in the region of the middle meatus may be troublesome; these granulations are best controlled with biting forceps and application of silver nitrate. It must have been the experience of many observers that this type of wound can be overtreated postoperatively.

It is important that patients who have undergone radical external operations on the frontal sinus should be observed from time to time for one or two years to insure the patency of the nasofrontal duct region, to eliminate the tendency for hyperplastic changes to occur in the mucous membrane of the nose in the region of the nasofrontal duct, and to remove accumulated secretions if cavitation of the frontal sinus persists.

Patients should be forewarned that, for the first year or two, when they are exposed to extreme degrees of temperature, par-

ticularly cold, some redness and swelling of the wound may occur. These signs disappear after the circulation of the parts has been reëstablished. Numbness, which results from severence of the supra-orbital nerve, disappears in the course of months. Acute reinfections of the nose may occur; at times this may cause swelling of the soft parts and distress in the region of the nasofrontal duct. The application of heat and inflation of the frontal sinus will relieve these symptoms quickly.

SUMMARY

I have expressed certain personal opinions regarding the choice of external operation on the frontal sinus. These are based on the anatomic characteristics of the sinus and the pathologic nature of the disease. I have outlined the post-operative care which I have found useful.

In general, it can be said that well directed external operations on the frontal sinuses produce excellent clinical results and that the fear of disfiguring deformity of the forehead is of minor consideration. Excellent results have come about because surgeons are familiar with the technical improvements, including methods for anesthesia suggested by various observers, understand thoroughly the pathologic characteristics of diseased conditions common to the frontal sinus, and know the indications for the use of an external operation.

REFERENCES

1. Furstenberg, A. C. Osteomyelitis of the skull; osteogenetic processes in the repair of cranial defects. *Ann. Otol., Rhinol., & Laryng.*, 40: 996–1012 (Dec.) 1931.
2. Jansen, A. Zur Eröffnung der Nebenhöhle der Nase bei chronischer Eiterung. *Arch. f. Laryngol. u. Rhinol.*, 1: 135–157, 1894.
3. Küster, E. Ueber die Grundsätze der Behandlung von Eiterungen in starrwandigen Höhlen, mit besonderer Berücksichtigung des Empyems der Pleura. *Deutsche med. Wchnschr.*, 15: 233–236 (March 21) 1889.
4. Lillie, H. I., and Anderson, C. M. The two-stage operation for suppurative frontal sinusitis with external manifestations. *Arch. Otolaryng.*, 5: 152–161 (Feb.) 1927.
5. Lillie, H. I., and Williams, H. L. The external fronto-ethmo-sphenoid operation; a critical review of the literature and details of the technic in use at the Mayo Clinic. *Minnesota Med.*, 18: 786 (Dec.) 1935.
6. Lynch, R. C. The technic of a radical frontal sinus operation which has given me the best results. *Laryngoscope*, 31: 1–5 (Jan.) 1921.
7. Mosher, H. P., and Judd, D. K. An analysis of seven cases of osteomyelitis of the frontal bone complicating frontal sinusitis. *Laryngoscope*, 43: 153–212 (March) 1933.
8. Pastore, P. N., and Williams, H. L. Osteomyelitis of the frontal bone secondary to suppurative disease of the frontal sinus: report of a case. *Proc. Staff Meet., Mayo Clin.*, 13: 7–10 (Jan. 5) 1938.
9. Sewall, E. C. External operation on the ethmo-sphenofrontal group of sinuses under local anesthesia. *Arch. Otolaryng.*, 4: 377–411 (Nov.) 1926.
10. Skillern, S. R. Obliterative frontal sinusitis. *Arch. Otolaryng.*, 23: 267–283 (March) 1936.
11. Smith, Ferris. Management of chronic sinus disease. *Arch. Otolaryng.*, 19: 157–171 (Feb.) 1934

DEEP ABSCESS OF THE NECK

SURGICAL TREATMENT

JOHN F. BARNHILL, M.D.

Professor Emeritus of Surgery of the Head and Neck, Indiana University School of Medicine

MIAMI BEACH, FLORIDA

GENERAL REMARKS

DEEP abscess of the neck has always been looked upon as a serious and often fatal affection. Located as such an abscess always is in the midst of large arteries, veins, and nerves, the affection could scarcely be otherwise than dangerous. It may result from traumatism of the structures of the neck or from primary infection of tissues or cavities in the immediate environment, chiefly of the head. Stab wounds are especially apt to be followed by a deep abscess. But by far the greater number of deep cervical abscesses occur secondary to infection and suppuration of the accessary nasal sinuses, to tonsillar inflammations, to decay of the teeth or alveolar processes or to ulceration of the mouth and throat. (Fig. 1.) The more serious abscesses are secondary to streptococcic infection of the primary sources. Other types of bacteria are found less often.

It is a notable fact that a vast majority of all cervical abscesses are located in the anterior portion of the neck, the reason being that all the primary sources of the infection drain into structures that lie anterior to the bodies of the cervical vertebrae. Abscesses of the anterior and posterior portions of the neck are, therefore, different in most respects, the posterior infection and suppuration being due almost entirely to boils and carbuncles or to traumatic injury. Abscess of the anterior cervical region has a tendency to spread while involvement of the posterior segment does not. The reason is that the portion of the neck lying anterior to the cervical vertebrae contains several notable collections of loose connective tissue, favorable to suppuration, whereas that part posterior to the cervical spinal column has none. (Fig. 6.) Migration of an anterior abscess occurs in many instances, and especially in neglected cases, whereas change of position is rare and never expected in the posterior cervical region.

DEEP ABSCESS AND CERVICAL FASCIA

The arrangement of the cervical fascia is also an important factor in determining both the location and migration of pus. The air tract, consisting of the larynx and trachea, and the food tract, made up from the lower pharynx and esophagus, pass downward through the fore part of the neck and each is inclosed by the branching, deep fascia. This arrangement of the fascia creates passageways not only for food and air, but also a sort of tube for the passage of the carotid artery, the jugular vein and the vagus nerve. The important point relating to the latter of these pathways is that it contains much loose areolar tissue. Infection from a primary, upper source, as for example a decaying tooth or suppurating sinus, gravitates downward into one of the areas of connective tissue, inflammation and abscess quickly follow and, unless evacuated or walled off by inflammation, the abscess may sag down to a new location and may even reach the mediastinum.

CONNECTIVE TISSUE POCKETS[1]

Mention has already been made of definite collections of connective tissue in

[1] The author's views on operative measures in deep abscess of the neck make necessary a very brief preliminary discussion of the cervical fascia and of the location and nature of certain pockets of loose areolar tissue in one or another of which the abscess almost always forms.

the anterior portion of the neck. There are five such pockets, and since the deep abscess forms in one or another of these, it is important to know the exact location of each of them. It should first be stated, however, that no such thing as a pocket actually exists normally in the neck since the so-called pocket is filled with loose connective tissue. When infected from some primary source this connective tissue breaks down; its place is taken by pus and thus a cavity is formed in the spot where formerly the connective tissue was found.

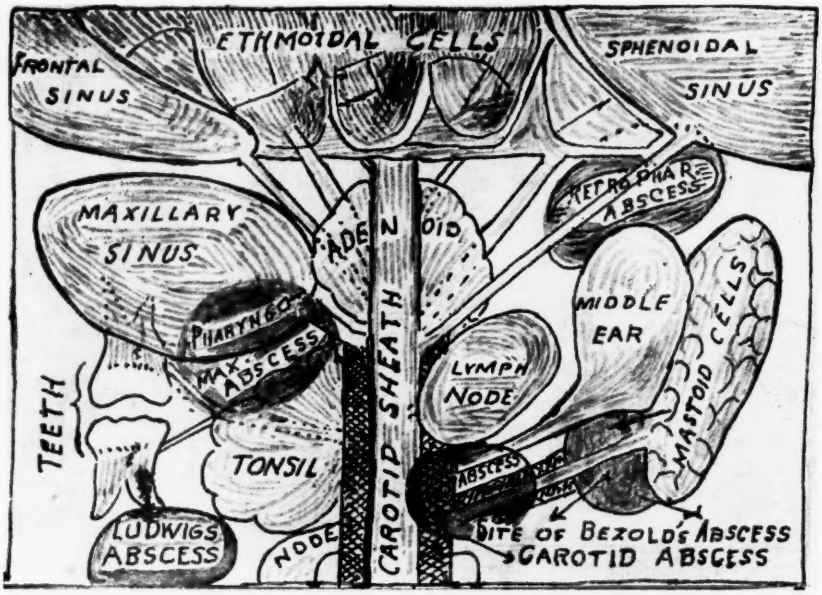


FIG. 1. Diagram showing the primary sites of infection leading to deep abscess of the neck. These are: the accessory nasal sinuses, adenoids, tonsils, ears, mastoid cells, lymph nodes and teeth. Infection in any of these may lead to abscess around the carotid sheath. Primary abscess of the neck probably never occurs. Note that all primary sources of infection drain directly into the connective tissue which surrounds the carotid sheath. Seats of primary infection have been shaded light, and abscesses shaded dark.

Of these five pockets the *first* lies in the floor of the mouth. The connective tissue here is abundant and binds loosely together the muscles that make the floor. Postgraduate students who dissect this floor for the first time invariably make some remark about the tissue—it is so striking. "How wonderfully beautiful and abundant it is," is a common exclamation.

When it is infected and abscessed, the affection in this pocket is commonly called *Ludwig's angina*. The abscess may spread rapidly. The affection is dangerous, the mortality being about 50 per cent.

The *second* connective tissue pocket lies at the top of the nasopharynx between the mucosa and the muscles which rest upon the bodies of the upper cervical vertebrae. The connective tissue is so abundant here that the prevertebral fascia may be easily stripped away by any blunt instrument inserted beneath it, even by the finger. In this location it should scarcely be designated a pocket since it extends downward for a long distance, forming a sort of elongated bag.

A *third* area in which connective tissue abounds and deep abscess is found lies in the extreme upper portion of the neck just external to the faucial tonsil. Like the others already mentioned it is nothing more than a space which is filled with loose areolar tissue and is in no sense a cavity until this loose tissue becomes infected, suppurates and is destroyed. In the end a collection of pus occupies the site formerly occupied by the connective tissue. The

cavity thus created is limited internally by the buccopharyngeal fascia; above by the external surface of the base of the skull: Externally by the internal pterygoid muscle and the stylomandibular ligament. The superior constrictor muscle and the faucial tonsil separate it from the pharynx. (Fig. 3.)

Fig. 2. Cross section of mouth showing especially structures which make the floor. The deep fascia which has much surgical importance is well shown. The position of the abscess (Ludwig's angina, 8) is shown in an early stage. Note that it is then separated from the oral cavity only by the mucous membrane. It may later descend through the strong fascia and mylohyoid muscle (dotted circle) and may then be felt as a hardened mass beneath the jaw. Fluctuation at any stage is very rare. 1, cavity of mouth. 2, mucosa covering tongue. 3, bicuspid teeth. 4, sublingual glands. Note that the abscess has compressed gland on right. 5, section of genioglossus muscle. 6, section of geniohyoid muscle. 7, section of mandible. 8, section of submaxillary salivary gland. 9, section of anterior belly of digastric muscle. 10, mylohyoid muscle showing deep fascia. 11, right and left halves of tongue. Note aponeurotic midline of separation of two halves. 12, position of abscess. Note origin in decaying tooth.

The carotid sheath, which contains the internal carotid artery, internal jugular vein and vagus nerve, lies immediately behind. The tip of the styloid process approaches and lies near the posterior wall. The parotid and submaxillary glands lie right and left below. This pocket is called the *pharyngomaxillary space.* (Figs. 3 and 4.)

The *fourth* pocket also contains considerable loose areolar tissue. Like that which lies in front of the bodies of the cervical vertebrae, it is an elongated sack, rather than a pocket. It begins at the base of the skull and follows the carotid sheath into the mediastinum. (Fig. 1.) An abscess in this location may be secondary to infection and suppuration of one or more deep cervical lymph nodes which normally lie upon the carotid sheath; or (and this is more commonly the case) it follows burrowing of the pus to the sheath from some position higher up in the neck from which

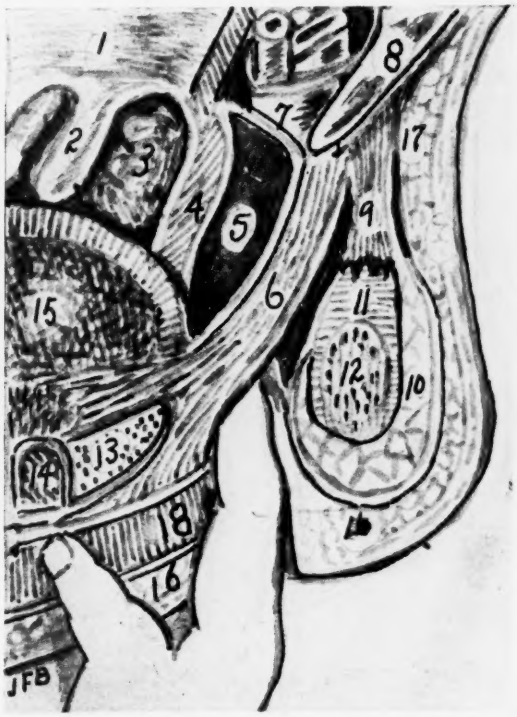


Fig. 3. Cross section of head showing position and relations of pharyngomaxillary abscess. 1, soft palate. 2, uvula. 3, tonsil. 4, superior constrictor muscle. 5, the abscess. 6, styloglossus muscle. 7, carotid sheath with common carotid artery, internal jugular vein and vagus nerve. 8, styloid process. 9, stylomandibular ligament. 10, parotid gland. 11, section of the ramus of the mandible. The upper end has been cut away to show the ligament. 12, section of body of mandible. 13, sublingual gland. 14, geniohyoid muscle. 15, tongue. 16, 17, skin and superficial fascia. 18, mylohyoid muscle. Note the strong layers of fascia. Note the position of the finger entering the abscess, and also that the superior constrictor muscle and its fascia are all that separates the abscess from the pharynx.

it has migrated. (Figs. 1 and 3.) For example, an abscess which begins in the nasal accessory sinuses, tonsils or adenoid, if neglected, may break through its walls and gravitate downward to a new situation anywhere along the carotid sheath.

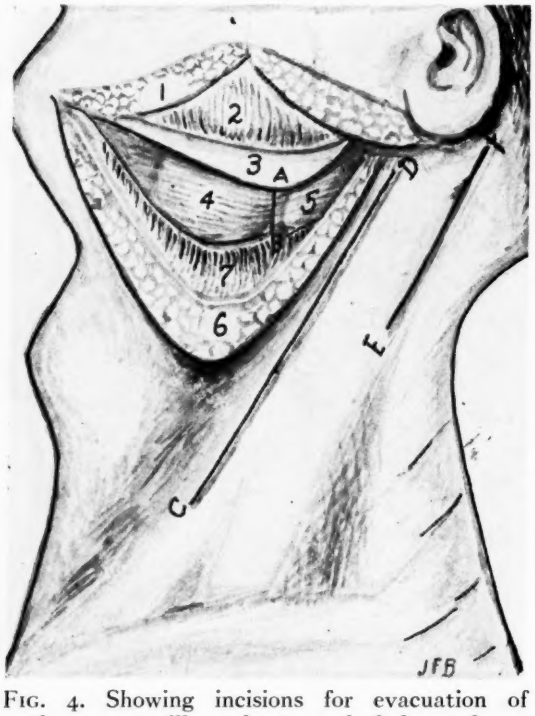


FIG. 4. Showing incisions for evacuation of pharyngomaxillary abscess and of abscess lower down around the carotid sheath. 1, 2, flap turned up. 3, mandible covered by periosteum. 4, submaxillary gland. 5, parotid gland. Note the thick, deep cervical fascia which covers these glands. 6, 7, lower flap. C_1-D, line of incision along the anterior border of the sternomastoid muscle. All these incisions go deeply to the external layer of the deep cervical fascia. A_1-B, line of incision through fascia between submaxillary and parotid glands. This opens passage directly to abscess. E-F, line of incision along posterior border of sternomastoid to reach Bezold's abscess.

A *fifth* pocket of loose areolar tissue is found just below the mastoid tip and along the posterior belly of the digastric muscle. The abscess which forms here originates in suppurating mastoid cells. It is nearly always found in cases of mastoiditis, operation on which has been neglected until the thin mastoid cortex has necrosed and ruptured and has allowed an escape of pus into the digastric fossa. The rupture is directly into the neck and the abscess lies deep to the sternomastoid muscle. The affection is called *Bezold's abscess* and should be distinguished from a postaural abscess which follows rupture through the mastoid process higher up and is in no sense a deep cervical abscess. In Bezold's abscess there is swelling, hardening and tenderness just below the mastoid tip. The neck is stiff and the patient turns his head with difficulty. There is much pain on deep pressure over the mastoid tip.

A deep cervical abscess may form in any one of the five pockets mentioned here. Abscess in any other place is rare and is the result of migration from the original site. Treatment of each requires a somewhat different operative technique.

COMPLICATIONS OF THE SEVERAL TYPES OF DEEP ABSCESS

Considering the compact and intricate nature of the anatomy of the neck it would be expected that structures adjacent to the abscess would become involved. In Ludwig's angina the swelling of the structures of the floor of the mouth is sometimes so sudden and violent that the tongue is crowded into the roof and the root of the tongue is pushed backward over the glottis. Impaired respiration from mechanical reasons may follow or difficult breathing may be due to extension of the swelling to the larynx when the patient, if not promptly relieved, may die of suffocation.

In every instance of deep abscess of the neck, and especially if the abscess is located along the carotid sheath, infection and thrombosis of the internal jugular vein or its branches may take place. While it is true that the abscess does not lie in actual contact with the veins, it is yet near enough to involve them in the septic process. Figure 3 shows the carotid sheath lying just behind the pharyngomaxillary cavity. In neglected cases instances have been reported in which the wall of the carotid sheath has become necrotic from the pressure of the abscess, the walls of the internal jugular vein, or even of the carotid artery, have ruptured, and fatal

hemorrhage has promptly followed. It will be seen, therefore, that the complications arising from deep abscess of the neck are of a serious nature and require immediate, skilful treatment to preserve the patient's life.

SURGICAL TREATMENT

Every deep abscess of the neck is a case for surgery. Medicines, poultices, and other general or local treatment are soothing but never curative and hence should not be long continued. To depend upon them to cure is deceptive and their continued use only invites complications of a dangerous nature to arise. Deep abscess of the neck is no place for the procrastinating surgeon. Pus when once formed here must be evacuated.

When to Operate. On this point there should be but little argument. It is almost universally conceded that the operation should be performed as soon as it is reasonably certain that an abscess is present. Some surgeons advise waiting until the abscess is walled off and thus avoid, as they believe, spread of the affection by the operation. Abscess of the neck has a natural tendency to wall off and hence by the time the diagnosis has been made it may usually be safely assumed that it is already well isolated. Again, some wish to wait until fluctuation can be made out and other trustworthy symptoms are present. Waiting for fluctuation is unwise and dangerous since this is always a late symptom. It will be recalled that the abscess is contained in a pocket of strong, deeply situated cervical fascia and that this pocket itself lies within a still stronger, more resisting external layer of cervical fascia. Considering these and other barriers, such as overlying muscles and skin, it is easy to understand that considerable time is necessary for the pus to necrose its way through and approach the surface. Meanwhile, as previously stated, the abscess may descend to the carotid sheath where its close proximity to the internal jugular vein and its large tributaries may set up infection and thrombosis of these vessels. Rupture of a deep abscess outward upon the skin surface is exceedingly rare but rupture of a pharyngomaxillary abscess into the throat is occasionally seen. A few cases of rupture of the abscess into the trachea or esophagus have been reported.

The surgeon who is familiar with the arrangement of the fascia and the distribution of the areolar tissue of the neck finds it comparatively easy and safe to cut down upon the suppurating cavity and evacuate the pus. He knows that the abscess is located in one of five very definite situations and hence he does not go blundering into all parts of the neck to find it. These several situations have already been discussed. The symptoms and signs commonly present point almost unerringly to the location of the affected pocket. Although the technique differs, the surgical principles involved in evacuating the pus are very similar in each instance. The surgeon never approaches the abscess through muscular structures since with very rare exceptions (traumatic) an abscess is never found inside a muscle.

OPERATION ON EACH INDIVIDUAL POCKET

1. *In Floor of the Mouth: Ludwig's Angina.* In the early stage the abscess lies just beneath the mucosa of the floor of the mouth and between the tongue and inner surface of the mandible. (Fig. 2.) A free incision, operating through the mouth, may prove sufficient and should be tried. An external operation may subsequently be performed if necessary. In a later stage the abscess probably will have found a lower level and will then lie upon the mylohyoid muscle. A strong layer of fascia and the muscle, skin, and fat separate it from the outside where it may show as a sagging tumor and be felt as a hardened, board-like mass. It is then best operated on by a free external incision. (Fig. 5.)

1. Make an incision from the angle of the mandible forward just inside the curvature of the jaw, almost to the symphysis. This incision should expose the strong fascia

shown in Figure 5. One or more large veins usually cross this field and should be ligated.

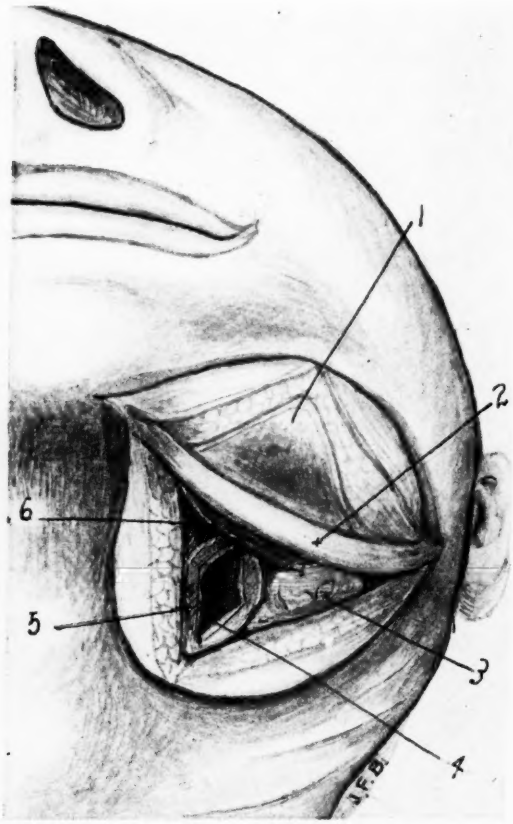


Fig. 5. Showing incisions for entering abscess in the floor of the mouth (Ludwig's angina). The incisions are longer than necessary, being made so to show better the structures involved. 1, upper flap. 2, mandible with periosteal covering. 3, submaxillary gland covered by external layer of deep cervical fascia. 4, opening through fascia and mylohyoid muscle. 5, mylohyoid muscle cut through. 6, mylohyoid muscle from which thick fascia has been incised and reflected.

2. Incise the fascia and mylohyoid muscle.

3. Insert a blunt instrument through the opening, in front of the submaxillary salivary gland and pass it upward toward the floor of the mouth. In the event the mouth can be opened widely enough, insert an index finger alongside the tongue. On passing the blunt instrument deeply enough through the outside opening its end may be felt by the finger inside the mouth. The mucosa should not be perforated, since if this be done, infection would probably enter from the mouth.

4. Insert a blunt-nosed forceps through the outside wound, and, separating the jaws widely, enlarge the incision through the fascia and the mylohyoid muscle. Sometimes the finger instead of the forceps may be entered to advantage.

5. Insert a fold of rubber tissue to the bottom of the cavity.

6. Partly close the primary wound around the drain. Provide for good drainage.

It will be noted that no large vessels or important structures of any kind will likely be injured by following the pathways shown in the illustrations. (Figs. 3 and 5.) Free hemorrhage from injury to branches of the anterior facial veins may be expected. Ligation of these should be at once performed. One must make certain of good drainage while healing progresses.

Clinical Example. Male, age 24, had been to a dentist a week previously for the extraction of a lower decayed tooth. The dentist told him the tooth was abscessed at the root. Three days later, the patient's temperature was 103 degrees, following a chill. He had severe pain in the floor of the right side of the mouth and opened his mouth with difficulty. The tongue lay to the left, apparently crowded firmly against the roof of the mouth. The mucosa in the floor of the mouth was edematous. Palpation on the affected side between the margin of the tongue and the mandible caused severe pain. Some swelling appeared under the jaw of the right side. The area felt hard and board-like. Swallowing was difficult and respiration slightly impeded. The leucocyte count was 10,500. The symptoms were worse twenty-four hours later and operation was urged.

The steps above described were followed, with operation as shown in Figure 5. There was much fat beneath the skin and so the mylohyoid muscle lay at great depth. No pus escaped after incision of the muscle, but when a blunt-nosed forceps was introduced deeply and the incision was spread widely, pus to the amount of an ounce welled freely from the wound. Recovery was uneventful.

11. *The Pharyngomaxillary Cavity.* Study of the anatomy of the region in

which this pocket lies shows that it is best opened from the outside. Reference to Figure 3, shows that it lies much nearer the pharynx than the skin surface, but to open the cavity from the pharynx, as may sometimes seem proper in the early stage of the abscess, leaves a pocket which drains badly and favors reformation of the abscess.

Entering from the outside would be at too great a risk unless the surgeon is familiar with the arrangement of the cervical fascia and its relation to vital structures. A study of this fascia shows that the cavity may be opened by the external route without serious risk by confining the procedure to a pathway that lies between the parotid gland posteriorly and the submaxillary salivary gland anteriorly. (Fig. 4.) The great vessels and nerves of the neck lie behind, near to but wholly outside of this pathway. (Fig. 3.)

Steps. 1. Make an incision from the middle of the submaxillary gland to the middle of the parotid (longer if necessary) following the curvature of the mandible and lying slightly below it. This incision should extend to the superficial layer of the deep fascia, which is dense over this area. The external facial artery and anterior facial vein often cross this field.

2. Ligate all bleeding vessels and cut through the fascia.

3. Insert a dull dissector inward and upward in the direction of the faucial tonsil. An index finger in the mouth placed just anterior to the faucial tonsil will readily detect the end of the approaching instrument. The path traversed by the instrument contains enough loose connective tissue to make the passage rather easy unless it has been inflamed, and hence force will not be necessary. The finger may sometimes be used in preference to an instrument. (Figs. 3 and 6.) When the finger has reached the bottom nothing separates the finger or instrument in the mouth from the finger in the pharyngomaxillary space except the superior constrictor muscle, its covering of mucosa inside and its buccopharyngeal fascia externally. It is sometimes desirable to incise these intervening structures and thus make a drainway from inside the mouth to the outer surface of the upper neck. Such an incision is especially indicated in those rare cases in which there is already a rupture of the abscess into the pharynx but with an inadequate opening for drainage.

4. Insert a drainage tube to the bottom of the abscess cavity. A drain made up of two or more folds of rubber tissue is preferable to one of plain gauze, or even to a cigarette drain. Gauze drains partially block drainage and are especially objectionable.

Each of the five locations where deep abscess of the neck is found is separate and different from the rest, presents its own problems and hence is separately described.

Clinical Examples. A female, age 35, had had both tonsils removed a few days previously, local anesthesia and snare being used. The operation was uneventful, but the patient did not do well following the operation. She remained in the hospital, in bed. A severe chill on the second day was followed by a temperature of 104°F. General sepsis was evident and there was much pain. Marked swelling of the tonsil area led to incision of the swelling with the evacuation of considerable bloody serum. Some relief from pain resulted, but the other symptoms became worse. The leucocyte count was 12,000, there was much prostration and repeated chills. The septic temperature continued. The incision was again explored, but was considered adequate. There was but little hardening of the structures at the angle of the jaw and, considering that there was but slight pocketing of the abscess which had ample drainage, no external operation was performed. Despite the fact that the abscess cavity was explored daily and sufficient provision for drainage was maintained, the patient died on the fourth day after the abscess was incised, the cause of death being apparently severe acute general infection.

Autopsy showed the lungs filled with small emboli and metastatic abscesses. The mediastinal and pericardial spaces revealed thin, slightly milky exudates, but only a very small amount of undrained pus was found in the

pharyngomaxillary space. No undrained, suppurating cavity of the neck was found. Infection of the pharyngomaxillary space at the time of the tonsil operation was assigned as the cause of the general sepsis and ultimate death.

A boy, age 18, had suffered much from one or more annual attacks of quinsy, the last one five days previous. He complained of much pain in the right side of the throat, difficult swallowing, respiration a little labored, and voice guttural. There was a great swelling in front of tonsil pocket, but no external tumefaction.

Incision of the pharyngeal swelling was followed by considerable discharge of pus and temporary relief from pain. The incision showed a tendency to close, but was reopened and the cavity explored. Pain returned, chiefly at the angle of the jaw, and there was a chill with rise of temperature to 103°F. Three days later there was swelling and hardness at the angle of the jaw and tumefaction increased. External operation was advised. The technique given in this article was followed, but in addition a long, curved blunt-nosed forceps was passed into the outer opening, through the cavity, through the former incision from the pharynx into the cavity and out into the pharynx. A thick fold of rubber tissue was grasped in the jaws of this forceps and was drawn out through the outer opening beneath the jaw, giving perfect through-and-through drainage. This drain was withdrawn a little each day and was finally taken out on the tenth day when healing was almost complete. Thorough drainage had accomplished a cure.

III. *Retropharyngeal Abscess.* There are two types of deep abscess found in the nasopharynx, each due to an entirely different cause. There are from one to three lymph nodes situated just beneath the prevertebral fascia in the upper pharynx. These are found almost exclusively in infants and very young children and are later atrophied. Because of their location they are called retropharyngeal nodes. Infection and breakdown of these glands ends in what is very properly called a retropharyngeal abscess. It is an acute affection which is accompanied by tumefaction of the nasopharynx, blocking of nasal respiration and difficult or impossible deglutition.

Both types of abscess of this region require free incision and good subsequent drainage; but the nature of each affection is such that a somewhat different technique should be followed.

1. *When due to suppurating retropharyngeal lymph glands,* the affection occurs in infancy or very early childhood. It is acute. The condition is serious; respiration may be so much impeded that tracheotomy must be performed without delay. Swelling of the nasopharynx is greatest on one side and extends below the free margin of the soft palate. Suffocation from sudden rupture of the abscess or from a sudden gush of pus following an incision into it may occur and hence the child's head must be in a hanging position while the examination or operation proceeds. A perfect-working suction apparatus should be at hand to remove the pus. A free incision performed through the mouth is usually all that is necessary.

2. In *Pott's disease,* the affection is chronic. No emergency usually exists. When respiration or deglutition becomes more or less seriously impaired, incision of the abscess may be made as above described. Evacuation of the pus usually removes the immediate distressing symptoms, but the affection is tuberculous and cure depends upon treatment by medicines, diet and prolonged residence in a suitable climate. An external incision is sometimes necessary.

Clinical Examples. A child, age 14 months, had had a severe head cold resembling influenza. He looked seriously ill, could not swallow and refused the breast. He was very restless and cried almost constantly. The respiration was labored, voice guttural, and temperature 103°F. Examination showed a large red tumefaction on the left side of the throat. The nasopharynx was blocked, and the soft palate and uvula pushed to the opposite side. No external swelling was apparent. Retropharyngeal abscess due to disintegration of retropharyngeal lymph nodes was diagnosed and a free incision of the abscess with the head in a hanging position was done. Suction apparatus was used to remove pus. Slow recovery

followed, owing to the physical condition of the child.

Pott's Disease. A boy of 19, who had never been strong came of a tuberculous family. One brother and one sister had died of tuberculosis. The patient had palpable cervical lymph nodes, and one had broken down and had been incised (scrofulous). No active tubercle of the lungs was present.

Present complaints were moderate pain and difficult swallowing. Fluctuation could be made out below the soft palate. The head was placed in hanging position, and the abscess was incised. A free flow of pus followed and the local symptoms subsided. The patient was sent to a tuberculosis hospital where much general improvement has since been made. Continued spondylitis has resulted in moderate kyphosis.

IV. *Deep Abscess along the Carotid Sheath.* Suppuration along this great pathway in a large percentage of cases is secondary to abscess in the head or neck higher up. It is also secondary when it follows traumatic injury or when it is caused by infection and disintegration of one or more of the deep lymph nodes which lie upon the carotid sheath. Primarily in many cases the abscess was in the pharyngomaxillary space, or was, perhaps, a Bezold's abscess that had migrated into its new position alongside the great vessels and nerves. It is in this new situation of the abscess that infection and thrombosis of the internal jugular vein is most apt to take place.

Symptoms due solely to the presence of pus in the pharyngomaxillary space are often so severe and so similar to the symptoms of thrombosis of the veins, that the surgeon may overlook the complication and so may put off the operation until the abscess has done serious damage. The internal jugular vein may be blocked by a septic clot; absorption into the blood stream may occur on the spot, or emboli may be carried to distant structures from which the infective material is distributed. It is evident, therefore, that a deep abscess in this location should be thoroughly evacuated as soon as its presence can be determined. Like abscesses in pockets previously described, this one is situated in the midst of numerous important structures, injury to which may result in danger or even the death of the patient.

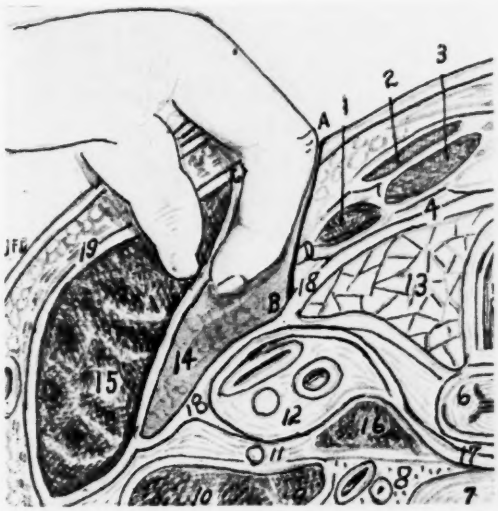


Fig. 6. Cross section of neck showing abscess (8) along the carotid sheath at level of first tracheal ring. Line A-B shows incision along anterior margin of sternomastoid muscle (line C-D in Fig. 4). This incision penetrates the superficial layer of the deep fascia at the point of union of the layer which passes above and the one below the muscle, C and D. The abscess is entered immediately after this fascia is cut through. The illustration shows the overlying muscle being elevated from the abscess. 1, 2, 3, ribbon muscles. 4, pretracheal fascia. 5, trachea. 6, esophagus. 7, body of cervical vertebra. 8, vertebral vessels. 9, 10, muscle. 11, connective tissue. 12, carotid sheath. 13, lobe of thyroid gland. 14, abscess. 15, sternomastoid muscle. 16, longus colli muscle. 17, 18, 19, deep cervical fascia. Note that the superficial layer of the deep cervical fascia is continuous with C at D and that after enveloping the ribbon muscles, it becomes the pretracheal fascia. Note also that as this fascia continues deeply into the neck it surrounds and protects the great vessels of the neck. Note that the abscess (14) lies in contact with this fascia, which forms the carotid sheath. Pus here under pressure may readily set up infection and thrombosis of the internal jugular vein.

Operative Steps. 1. Make an incision in front of the sternomastoid muscle from the tip of the mastoid process to about the third tracheal ring. (Line A, B, Fig. 4.) The length should be greater in cases in which the abscess has sagged to a lower level. Expose the superficial layer of the deep cervical fascia and cut through it along the

anterior edge of the sternomastoid muscle. (Fig. 6.) It is important that this incision be made far enough forward to divide the fascia after the two layers have passed around the muscle and have again united, for it is a surgical error to cut through the muscle even though that course be shorter and straighter. (Fig. 6.) Retract the sternomastoid muscle from the carotid sheath.

2. To do this, follow the deep layer of fascia which passes under the muscle, using a blunt instrument or even the finger to make the dissection. (Fig. 6.) The muscle is normally connected to the carotid sheath by loose areolar tissue and since this tissue has been destroyed by inflammation and the abscess has taken its place, the dissection is easily made. Since the abscess lies just outside the carotid sheath, the pocket will be entered, and the pus evacuated as soon as the sheath is reached. (Fig. 6.) In event there has been considerable inflammation in the region of the abscess, it may be found impossible to follow the planes of fascia. It will then be necessary for the surgeon to use his anatomic knowledge and skill and to enter the cavity somewhat blindly—at least without good landmarks as guides.

It is found occasionally that the deep cervical glands are infected and greatly enlarged, so that they must be dealt with. Sometimes the surgeon finds it better to enter the cavity from the posterior edge of the sternomastoid muscle (line C-D, Fig. 4) in which case he should bear in mind that the spinal accessory nerve passes from beneath the muscle at the junction of the upper and middle thirds, and that to injure it will result in serious impairment of motion of the shoulder.

3. Insert a soft rubber drain deeply into the abscess cavity and, 4, partially close the wound around the drain. Maintain good drainage until healing is complete.

Clinical Example. A female, aged 24, about three weeks previous had had a sore throat which her physician thought might have been quinsy. There was improvement but not cure from local medication. A few days later there was pain, swelling and hardness at the angle of the jaw, which improved after poulticing. Almost three weeks from the onset the neck of the affected side was swollen and hard from the angle of the jaw to below the level of the cricoid cartilage. There was much stiffness of neck, slight rise of temperature and pain on palpation, but no fluctuation. The leucocyte count was 15,500.

A deep cervical abscess along the carotid sheath was diagnosed. It was believed the infection had passed from the tonsil to the pharyngomaxillary space and then gravitated from the space to the upper portion of the carotid sheath.

The external operation was done, following steps outlined above. Primary incision was made along line A-B. (Fig. 4.) The deep lymph nodes were enlarged but not broken down and a large abscess was present. Recovery was prompt.

v. *Bezold's Abscess.* 1. In the event that the simple mastoid operation has not already been done, perform it now as a necessary step to reach the abscess.

2. Remove the mastoid tip completely. The rupture through the cortex of the process which has permitted escape of pus deeply into the neck has occurred into the digastric fossa on the inner surface of the tip and hence the best way to reach it is to completely remove the overlying bone. In most instances the removal of the mastoid tip opens the abscess widely and permits escape of the pent-up pus. It should be recalled that the pus lies deep to the insertion of the sternomastoid muscle which must, of course, be detached from the mastoid process.

3. Insert a probe into the abscess cavity which lies beneath the sternomastoid muscle and pass it downward to the bottom. In neglected cases the pus may have dissected its way, first along the posterior belly of the digastric muscle and then along the carotid sheath, for a considerable distance. With the probe inserted to the bottom of the cavity and with the free hand over the outside of the neck, the tip of the instrument can usually be felt posterior

to the posterior border of the sternomastoid muscle.

4. Cut down upon the deep end of the probe and pass the latter out through the opening.

5. Enlarge the lower opening to a considerable width. In some cases it will be best to connect the upper and lower openings by a deep incision and thus lay the entire cavity widely open. Should this step be carried out the incision should be made along the posterior border of the sternomastoid muscle. (Line C-D, Fig. 4.)

6. If this is done pack the wound loosely and treat it subsequently as an open cavity.

7. Should an upper and lower opening be considered sufficient, pass a good sized fold of rubber tissue through the abscess pocket by means of a curved dressing forceps and permit an extension of an end of the drain from both the upper and lower openings.

8. Pack the mastoid wound separately, after closing only the upper end.

9. Place a large thick pad of gauze over each wound and apply bandages.

Clinical Example. Male, age 65, had been ill for three months following influenza and middle ear abscess. The drumhead had been incised, but the ear continued to discharge and there was much pain in the region of the ear and mastoid, especially at night, with no tendency to recover. Examination at the end of three months showed the patient seriously ill. He had lost weight, felt weak, slept poorly, and was in constant pain. A profuse discharge from the ear had been present until a week before when it had stopped. Pain and stiffness of the neck followed cessation of the discharge, and the patient could not turn his head because of the stiff neck. Pain on pressure was experienced over tip of mastoid process and upper neck; this had been called rheumatism of the neck. Temperature was 99. White blood count was 10,000. Perforation of drum head produced no discharge. Moderate swelling and board-like hardness were noted over the neck near the mastoid process. The diagnosis was Bezold's abscess following a rupture of the mastoid cortex, allowing escape of pus into the digastric fossa.

Complete, simple mastoidectomy was done and the entire mastoid tip removed. A large necrotic opening leading into the digastric fossa was found. Much pus escaped under pressure. A probe could be passed beneath the sternomastoid muscle for a distance of 8 cm. The lower end of the probe was cut down upon; curved forceps were passed in at the upper opening and out through lower. The jaws of the forceps were opened widely to spread the lower opening and to insure adequate drainage. A corrugated, soft rubber drainage tube was passed in at the upper opening and out at the lower. The mastoid wound and the wound in the neck were dressed separately. Recovery was slow but complete.

Surgery of the Complications of Deep Cervical Abscess. Each of the five abscesses above described has its own special complications and likewise its own requirements for surgical treatment. Abscess in any situation is sometimes complicated by serious general infection. The swelling of the mucosa of the floor of the mouth in Ludwig's may extend suddenly to the larynx, when the patient will be threatened by suffocation, so alarming that an immediate tracheotomy must be performed. Obstruction of the larynx may also occur in pharyngomaxillary abscess.

One of the most serious complications arising from retropharyngeal abscess is edema of the glottis, which also may demand early tracheotomy. Another danger arises from sudden rupture of the abscess, perhaps, during sleep, with a gush of pus into the throat sufficient to suffocate the patient.

The complications arising from pharyngomaxillary abscess and abscess lower in the neck, along the carotid sheath, are very similar. Infection and thrombosis of adjacent veins occur oftenest and prove most serious. Figure 3 shows the close proximity of the internal jugular vein and its large tributaries to the abscess, and clinical experience has shown that these vessels are not infrequently infected in the immediate presence of suppuration. The pterygoid plexus of veins lies quite close to the seat of a pharyngomaxillary abscess

and may also be secondarily affected (thrombosed).

Infection and thrombosis of the extreme upper portion of the internal jugular vein is the most serious complication of Bezold's abscess. This large vein as it emerges from the skull lies just inward and forward of this abscess. Pus in this location, confined as it is by powerful muscles and strong fascia, favors entrance into the blood stream of the infected material. Evidence of involvement of the veins is usually present: a chill, high temperature, sweat, prostration and high leucocyte count announce the onset of the complication. Operation which includes the removal, when possible, of all portions of the infected, thrombosed veins is urgent in every case.

Steps. 1. Transfusion as a preparatory measure when the condition of the patient indicates.

2. If the deep abscess has not already been opened and drained, carry out this procedure by following the steps already mentioned.

3. Open the carotid sheath, isolate the internal jugular vein and inspect it with care. If thrombosed, determine the limits of the clot.

4. Ligate the vessel above and below the clot.

5. Excise and remove the diseased portion of the vessel. Examine the tributaries, and especially the facial vein.

6. Should it be thrombosed, ligate and excise the affected part. In event the thrombus in the internal jugular vein lies wholly above the entrance of the facial vein, and the latter continues to function, it should not be ligated. It is one of the main emissaries from the brain and to close it when the internal jugular has only just been resected may so seriously retard intracranial circulation as to sacrifice the patient's life.

7. Pack the wound loosely with gauze which is to be changed as often as soiled. Until the intracranial circulation has adjusted itself to the status of the crippled return circulation through the emissary veins, the patient should be kept quiet both physically and mentally.

Clinical Example. Male, age 21, had been ill for eight weeks following a severe head cold which began during an epidemic of influenza. He consulted no rhinologist, but the symptoms seemed to suggest a serious inflammation of the nasal mucosa which extended into the nasopharynx, pharynx and possible the nasal accessory sinuses. The patient developed a deep-seated swelling on the right side near the angle of the jaw. This was very painful and poultices were applied on his doctor's orders. No tendency to recovery was manifested. Chilly sensations were followed by fever, the neck became increasingly stiffer, and the patient felt very ill.

On examination six weeks after onset, the patient appeared quite ill. Following a severe chill, his temperature had risen to 103°F. Profuse sweating, increased pain and stiffness of the neck were present. Leucocyte count was 18,500. The neck was swollen over the upper right side and felt hard and board-like on palpation. There was no fluctuation. A severe chill on the following day was accompanied by a leucocyte count of 20,000. The temperature began a saw tooth tracing.

Deep cervical abscess with infection of the internal jugular vein was diagnosed.

A transfusion was given. Following the operative steps outlined above, a large abscess lying between the carotid sheath and sternomastoid muscle was opened and drained. (Figs. 4 and 6.) The carotid sheath was then opened and the internal jugular vein was carefully examined. It was thrombosed to a point below the entrance of facial vein. The vessel was ligated and the thrombosed part exsected. A portion of the facial vein was also exsected. Two sutures were taken in the upper part of the wound. The lower part was left open and loosely packed.

The temperature dropped sharply in twenty-four hours, then slight rigors occurred, followed by a rise. White count was 18,500. Another transfusion was given, but the septic temperature continued. We removed the upper sutures and made a careful examination of the wound. Some enlarged deep lymph nodes were inspected but none were suppurating. Emboli had set up metastatic areas of infection which could not be reached by surgical means and

the patient rapidly sank, dying on the sixth day following operation. This was a badly neglected case and the prognosis was bad at the time of operation.

Until rather recently the mortality from deep abscess of the neck has usually been stated to be about 50 per cent. At present, however, it is much less. Improved statistics have been achieved chiefly through a better understanding of the cervical fascia and of the several pockets of loose connective tissue that lie within its folds. Earlier diagnosis and earlier operation on the abscess and for the complications which attend it, have also been important factors in the betterment of the statistics. Mortality should be reduced to 25 per cent, or even less.

THE implied contract created by the physician-patient relationship also places the physician under the obligation of continuing to render his services as long as the patient requires aid. . . . In other words, the patient has the advantage under the contract because he may discharge the physician without cause or explanation, while the doctor must abide by the wishes of the patient.

From—"The Roentgenologist in Court" by Samuel Wright Donaldson (Thomas).

ORAL LESIONS IN OTOLARYNGOLOGY

DEAN M. LIERLE, M.D. AND RUBEN NOMLAND

Department of Otolaryngology, University Hospital Department of Dermatology, University Hospital

IOWA CITY, IOWA

THE object of this paper is to summarize briefly and generally some of the available material relating to those lesions of the oral cavity which come frequently to the attention of the otolaryngologist and the general practitioner.

Early diagnosis of these lesions is highly important to physician and patient. If this presentation emphasizes such need, and in addition suggests further study, its purpose will have been accomplished. Naturally, limitation of space makes impossible detailed discussion of the diseases, and permits only the briefest suggestions regarding diagnosis and treatment. No attempt to be original has been made. Sources of the material offered are recognized published texts of specialists in the field, as well as the writers' own experience and observations. Those who wish to avail themselves of reliable reference books will find a number listed at the close of this article.

Many of the lesions of *syphilis* occur in the oral cavity and on the lips. The extragenital primary lesion is found most frequently on the lips, but occurs elsewhere in the buccal cavity. It begins as a small, eroded papule which may simulate a "cold sore" or other innocuous lesions. It enlarges to form a single firm lesion, usually eroded and crusted, but seldom ulcerated.

If it is uncomplicated, it is not painful, but it is larger than the ordinary genital chancre. Secondary infection with pain, ulceration and other evidences of inflammation are often present.

The regional lymph nodes are enlarged but not particularly painful unless there is secondary infection. Healing takes place in several weeks, though the induration may be present for months. Scarring occurs when the lesion has been secondarily infected.

The diagnosis is made on the finding of a single indurated lesion of several days' or weeks' duration, with marked regional adenopathy. The dark field examination is usually positive and the Wassermann becomes positive after the chancre has been present for two weeks. A primary lesion may be differentiated from an epithelioma, since the history of the latter is longer and there is no early involvement of the regional nodes. Biopsy and pathologic studies will confirm the diagnosis. A chancre also may be differentiated from herpes simplex, which has a short duration and shows groups of vesicles on an inflammatory base.

Oral lesions are common in papular *secondary syphilis*. They may, however, be the only secondary lesions present, and they are a common relapsing lesion. There are three types: the mucous patch, the split papule and the syphilitic angina.

Mucous patches appear as red, superficial, sharply outlined erosions of mucous membrane frequently covered by a white pellicle. They may localize on the tonsils, uvula, soft palate, buccal mucosa, sides of the tongue and inner surface of the lips. Numerous spirochetes are present in the lesions and they are highly infectious.

Split papules occur at the commissures of the mouth as red, eroded, elevated lesions on contiguous portions of the upper and lower lips. (Fig. 2.) They simulate perleche which is described later. Syphilitic angina often presents no lesion but has a diffuse redness of the tonsils and adjoining pharynx.

In most of the mucous membrane lesions of secondary syphilis there are other evidences of the disorder on the body and the Wassermann is always positive.

Late or *tertiary syphilis* appears many years after the original infection. The gumma is the most common lesion and may localize anywhere in the mouth, such

Fig. 1. Chancre of primary syphilis.

as on the tongue, hard palate, cheeks, and occasionally on the lips. It begins as a deep swelling and inflammation which erodes the overlying tissues to form a deep, irregular, sloughing ulcer. Gummata may be quite destructive and when they occur in the hard palate, a common location, they cause perforation. (Fig. 3.) Small gummas may occur in the tongue, but the most common lesion of the tongue is an interstitial inflammation with thickening of the tongue. This leads to atrophy of the tongue and its mucous membrane.

More superficial destructive lesions occasionally occur in the mouth and pharynx. These give rise to sclerosis and scarring, but have no true gumma formation.

Lesions of *congenital syphilis* are not different from those of acquired syphilis. Rhagades, scars and fissures, radiating from the angles of the mouth, and Hutchinson's teeth, are special lesions of congenital syphilis. Atresia of the pharynx occasionally may be found.

Many other lesions, such as pemphigus, impetigo, erythema multiforme, lichen planus, drug lesions and stomatitis must be differentiated from mucous patches.

Fig. 2. Split papule of congenital syphilis.

Tuberculosis and carcinomas also must be differentiated from tertiary lesions. Tuberculous ulcers are not so deep, and their edges are irregular. Laboratory tests and general examinations are helpful in making diagnoses. Carcinomas which may be associated with gummas, particularly in lesions of the tongue, can be differentiated by pathologic studies.

Leucoplakia is an inflammatory disease affecting the mucous membranes of the tongue, cheeks, gums, lips and palate. It is common among middle aged men but occasionally is found in younger male adults, and, rarely, in women. The belief by some that leucoplakia is syphilitic has not been proved.

The disease appears first as patchy redness upon the mucous membranes, changing to bluish tint and finally becoming white and shining, with a nodular surface. Smooth, white lines mark the tongue. Sometimes the mucous membrane of the oral cavity becomes wrinkled, with "hardened furrows." Unchecked progress of the lesion may result in malignancy. At all stages it responds reluctantly to treatment.

Its causes are not definitely established, but use of tobacco in excess is considered a

contributory factor. A burn on the tongue often develops into the shiny white patch characteristic of leucoplakia. Irritations

Fig. 3. Perforation of hard palate. Healed tertiary lesion.

from teeth, ill-fitting dentures, highly seasoned foods, alcoholic beverages or fillings in the teeth, are other causes of the lesion. Its recurrence is common, particularly if irritants are not eliminated permanently.

Leucoplakia is not confined to specific areas. Its appearance on the palate is characterized by enlarged mucous glands and gray-white surface. Patches in the cheeks usually are triangular in shape. When linear markings appear on the tongue, its surface is rough to the touch and the tongue is heavy. The raised plaque variety is identified by heavy white patches surrounded by normal tissue, on the tongue, cheeks, hard palate and lower gum surfaces. More serious is the papillomatous type which may result in leucoplasic fissures and erosions with cancerous tendencies. Carcinoma has a tendency to develop more readily when syphilis is a complication. Leucoplakic patches may be differentiated from syphilitic mucous patches, however, by the acute development of the latter, and also by other indications of syphilis.

At the outset, changes in the superficial

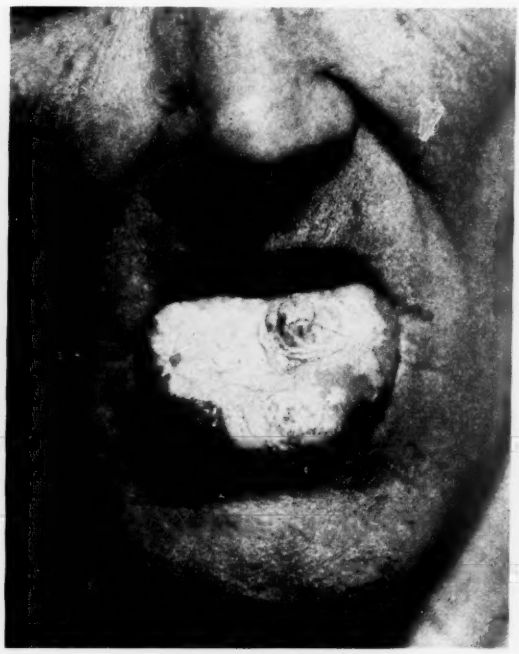


Fig. 4. Leucoplakia with epithelioma of tongue.

epithelium are characteristic. These may be followed by almost complete destruction of the papillary structure, and by changes in the submucous layers. Few diseases resemble this one, though mucous patches and lichen planus sometimes cause confusion. Serologic studies will aid in the identification of leucoplakia and if cancer is suspected, tissue should be examined histologically.

Removal of irritants and improvement of oral hygiene may be all the treatment necessary for early leucoplasic lesions, but in the more advanced cases this treatment may be augmented by cautery or surgical resection. Electrocoagulation, *x*-ray and radium treatments also have been advocated.

Lichen planus is an inflammatory skin disease of unknown etiology. In addition to the diagnostic, flat, angular, shiny papule found typically on the flexor aspect of the wrist, there also may be lesions on the buccal mucosa, gums, tongue, hard palate, uvula or lips. At the outset the papules are elevated and distinct, surrounded by normal mucous membrane,

but they may coalesce later, becoming smooth and flat. Sometimes they are linear, criss-crossing in a network on mucous membrane areas.

Fig. 5. Lichen planus.

When the lesions appear on the tongue, they vary in size and shape. Oval or circular plaques may appear on the back of the tongue, while on the upper and lower surfaces single papules or streaks are common. The streaks or networks are not often found on palates, gums or tonsils. Solitary lesions are most commonly seen on tonsils, and either solitary or aggregated papules in the other areas mentioned. Lichen planus stubbornly resists treatment and irritation is felt when spicy or pungent foods are eaten.

Lichen planus may resemble leucoplakia, but it differs by being less infiltrated and practically symptomless. Likened also to syphilitic mucous patches, these lesions differ by being surrounded by normal mucous membrane instead of a generally inflamed area.

The disease may disappear without treatment, or it may be treated with Fowler's solution, *x*-ray, or injections of neoarsphenamine, sodium cacodylate or mercuric chloride. Intramuscular injections of bismuth compound are sometimes beneficial.

Lymphangioma, caused by the dilatation of the lymph spaces of the tongue and lips, is another disease which should be diagnosed early. Neglect may result in deformities. These lesions, usually congenital, may remain stationary for years, only to develop into large cysts during adolescence or later years.

Fig. 6. Lymphangioma of tongue.

The disease begins with the enlargement of lymph spaces beneath the epithelium. Vesicles appear on the surface of the lips or tongue, which ooze lymph when ruptured or punctured. These vesicles may be so small as to escape detection, or they may be large, covering considerable area. Sometimes the entire tongue is involved. As the disease progresses, the tongue swells until it no longer can be held in the mouth. Ulcers form, a foul condition exists, and excessive salivation is common. Jaws and palate may be deformed, and teeth displaced. The treatment advocated is surgical excision, when possible. Irradiation and cauterization also are suggested.

There are two types of *hemangioma*, simple and cavernous, both congenital and of unknown etiology. They are reddish-purple, varying in size from eruptions of

pinpoint size to areas involving most of the lip, cheek and tongue. Common types are spongy, the large hemangioma being lobulated. They rarely ulcerate, but may become so large as to destroy tissue, because of the disturbances in the underlying blood supply. The most common sites for these lesions are the lips, tongue and cheeks. Usually they are painless, but the larger cavernous types are disfiguring and unsightly and interfere with functions.

Fig. 7. Cavernous hemangioma of lip. Before treatment.

Fig. 8. Same patient as in Figure 7. After treatment.

Radium and *x*-ray are used in the treatment of hemangioma. Electrocoagulation has been found effective for the cavernous types, several treatments usually being necessary.

Herpes simplex is believed to be caused by a filtrable virus. The disease often accompanies or follows gastrointestinal disturbances, head colds or general systemic disturbances such as fevers, pneumonia and sunburn. Recurrent attacks are believed to be related to focal infection or to some toxic condition in the gastrointestinal tract.

Very early a burning sensation is felt in an erythematous area. Later groups of very small vesicles appear, some of which later coalesce to form blebs. The vesicles are filled with a clear or hemorrhagic fluid and are usually in clusters. On the lips, the cutaneous surface later becomes yellowish and crusty, while in the mucous membrane there are areas of desquamation. The eruption lasts from five to six days to several weeks. After healing, a reddish area remains for some time. There usually is little pain, unless the vesicles become infected. Mucous membrane of the cheeks, tongue or palate may be involved as well as the lips. Lymph nodes also may be infected.

Treatment with local applications of oxyquinoline sulphate, 1:1000, administration of arsenic, *x*-ray, lotio alba, and alkaline mouth washes are suggested. Improvement of general health, correction of gastrointestinal disturbances and, in recurrent cases, the removal of the foci of infection, also are important.

Warts rarely involve the mucous membrane of the lip, tongue or cheek, and only occasionally are found in other parts of the mucous membrane of the oral pharynx. Infection is thought to be an etiologic factor. They are often confused with verrucous hyperkeratosis; this is potentially malignant and should be removed.

Carcinoma may involve the lips, tongue and any part of the mucous membrane of the mouth. It also may invade the jaw, or may arise from embryonic epithelial cell nests in the peridental membrane. Practically all carcinoma of the mouth is of the squamous cell type, but basal cell epitheliomata may also appear in the mucous membrane.

Leucoplakia, hyperkeratosis and fissures are definite precancerous lesions. Irritants such as alcohol, tobacco, betel nut, chemicals, oral sepsis, jagged teeth and syphilis are predisposing causes.

Although it may involve any part of the tongue, carcinoma most frequently is found in its lateral borders, the areas most frequently irritated. Cade and Spencer list carcinomas as ulcerative, papillary, nodular, fissured, atrophic and multiple. Early symptoms, other than the presence of the tumor, are few. Pain may be felt if the cancer is the ulcerative type. Late developments are immobility of the tongue, pain, hemorrhage, foul breath, glandular involvement, loss of weight and strength and secondary anemia. Diagnosis can be made only by histologic examination. The tumor must be differentiated from syphilitic, tubercular and benign lesions, and laboratory tests will aid in the diagnosis.

Neither time nor space will permit adequate discussion of treatment here. Irradiation, diathermy and surgery are types of treatment used most commonly. The type of treatment followed is dependent upon a number of factors, such as the general condition of the patient, the location and type of tumor, metastases, etc. Very often, a combination of all three types of treatment is necessary.

The prognosis in the treated cases varies according to the age of the patient, and to the condition, location and type of the tumor. Carcinoma of the lip and buccal mucous membrane offers a better prognosis than carcinoma of the tongue.

Fig. 9. Thrush.

Perleche is a simple erosion involving the mucous membrane of the angles of the mouth. The disease is contagious and occurs in epidemic form in families, schools or institutions, and is transmitted by contact, eating utensils and the like. It is caused by an aerobic streptococcus, or a yeast.

It is characterized by whitish membrane which starts at the angles of the mouth. Slight thickening and maceration occur, leaving superficial fissures, which, as a rule, do not bleed. The patient complains of a burning and dry sensation and constantly licks the lesions. There is no residual scarring.

Laboratory tests will differentiate the disease from the split papule of secondary syphilis. Perleche also may be confused

with herpes simplex, eczema, aphthae and stomatitis.

For treatment, Sutton and Sutton advise frequent applications of tincture of iodine. Silver nitrate, 10 per cent, and chromic acid in 2 or 3 per cent solution also are suggested.

Fig. 10. Fordyce's disease.

Thrush is an infection due to a yeast-like organism, Monilia albicans, which invades any part of the mucous membrane of the buccal cavity. It is most common among infants, though older persons in the late stages of such diseases as diabetes, tuberculosis, carcinoma, typhoid or pneumonia sometimes are affected.

Usual causes for the growth of the organism are faulty cleansing of babies' mouths after feeding; infection of nursing babies from mothers' breasts; contamination from unsterile bottles, water, formulae or rubber nipples, and infection from unsanitary nurseries. Neglect of mouth hygiene and lowered physical condition during wasting diseases are the most common causes of thrush in older people.

The disease first manifests itself in small white spots which appear in the mouth, growing and coalescing to form whitish patches. If these are scraped off, the surface beneath is red and raw. Large areas may be covered with this thick coating, resembling diphtheritic patches. Some pain is felt at the outset. If the infection is severe, a sweet, acid odor is present and the surface of the patches is slimy-white. Serious consequences may be expected if the thrush spreads to the bronchial or intestinal tracts.

Bacteriologic examination may be necessary to distinguish between thrush and such diseases as diphtheria, and mercurial, ulcerative or necrotic stomatitis. Non-irritating, alkaline mouthwashes and local applications of gentian violet are of value in treatment.

Trench mouth, also known as Vincent's angina, is an oral disease which has, at times, assumed epidemic proportions in barracks, prisons, asylums or orphanages. It is a highly infectious disease involving

the mucous membrane and lymphoid tissue or the oral pharynx, and usually is contracted by direct contact, or from use of infected eating or drinking utensils.

The organisms which cause trench mouth are fusiform bacillus and the spirochete of Vincent. They usually attack the mucous membrane of the alveolar process, faucial and lingual tonsils, mucous membrane around the teeth, and, less frequently, the mucous membrane of the tongue.

The disease is characterized by superficial irregular ulcers, covered by a dirty, grayish membrane which bleeds easily when removed. Congestion around the ulcerated areas is noted. Ulcerations of the faucial tonsils are seen frequently at the upper pole. At times, these ulcers are quite deep with some destruction of tissue. A fetid odor is apparent.

The regional lymph nodes are slightly enlarged and may be somewhat tender. Swallowing is painful and occasionally some slight systemic reactions may be observed.

Treatment consists of local applications of neoarsphenamine paste, or Fowler's solution. A 5 to 10 per cent solution of chromic acid also may be useful. Repeated small doses of intravenous neoarsphenamine sometimes aid cases which do not respond to local treatment. Hydrogen peroxide and sodium perborate also are suggested. Lesions about the teeth may be very resistant to treatment and may resemble pyorrhea alveolaris.

Actinomycosis, classed as a fungus infection, is particularly common in agricultural areas and more prevalent among men than women. Invasion by actinomyces, or ray fungus, is the cause of the disease. It usually gains entrance through the mouth, and the teeth, tonsils, tongue and jaws may be involved. It manifests itself with small, nodular swellings which later break down, discharging yellow, purulent liquid containing the ray fungus. Tiny sulphur granules are exuded. Healing in one place may be simultaneous with outbreak in another.

Bacteriologic and serologic studies will reveal the essential difference between these lesions and syphilis, carcinoma, tuberculosis, infected cysts or blastomycosis.

Fig. 11. Scrotal tongue.

Radical surgery, *x*-ray, iodides and copper sulphate solutions are a few means suggested for treatment.

Pemphigus is one of the most serious diseases affecting the buccal cavity. Oral lesions may occur before, during or after skin eruptions appear. The onset of the disease usually is insidious. Warning symptoms are pain in swallowing, dryness of the throat and a sensation of burning, especially while hot or spicy foods are being eaten.

Rounded blebs, varying in size from those of pinpoint proportions to others which may be centimeters in diameter, appear upon lips, soft palate, pharynx, or other areas of the oral mucous membrane. They grow rapidly, rupturing within a few hours or a few days of the onset of the lesions. A serous discharge, sometimes streaked with blood, is released when the blebs rupture. Pain increases with the rupture, and as the desquamated epithelium is detached by trauma, the area beneath appears red and eroded, and may present a red areola.

The older lesions, except in severe cases, tend to heal, only to be followed by a new crop. In more chronic cases, and those

which have been particularly limited to the mucous membrane, deep cicatrices and adhesions of the membrane may develop. Recurrent oral blebs and erosions, and persistent loss of weight are characteristic of the disease. When present only in the mucous membrane, diagnosis is not easy.

To distinguish pemphigus from secondary syphilis, serologic or therapeutic tests may be necessary. Vincent's angina, diphtheria, drug eruptions and erythema multiforme are other diseases with characteristics similar to pemphigus, but bacteriologic and histologic tests should readily differentiate between them. As to prognosis, recovery may take place after years, but the disease usually is ultimately fatal.

Improvement of general health by diet, blood transfusions, ultraviolet ray treatments, and internal administration of quinine are helpful in the treatment of pemphigus. Correction of oral hygiene is important, and intramuscular injections of coagulen and cacodylate on alternate days have been found beneficial. Acetarsone, by mouth and intravenously, and germanin also have been found successful.

Erythema exudativum multiforme appears in the oral cavity as acute inflammatory patches. Vesicles and blebs which itch, burn and pain are coincident with bullous skin lesions appearing characteristically on the dorsum of the hands. Occasionally the disease is found only in the oral pharynx. The mouth lesions are similar to mucous patches, stomatitis, or to pemphigus lesions. The early eruptions are vesicobullous in character, but the blisters soon rupture, leaving a painful eroded area. Attacks of erythema exudativum multiforme usually are of short duration. They are not serious, but usually are recurrent.

Dietary measures are important in the treatment of this disease. It may be possible, also, to discover and eliminate the foci of infection. Use of alkaline mouth washes and internal dosage of salicylates are found beneficial.

Periadenitis mucosa necrotica recurrens is a somewhat rare disease of the oral cavity. It also is known as chronic aphthae and is found more frequently in women than men. Its etiology is unknown. Histologically, sections reveal chronic inflammation and necrosis. Loblowitz suggests that the lesions may be angioneurotic in origin. Most of the cases we have seen occurred in women over forty years of age.

The disease begins with small nodules which involve the mucous membrane of the cheek, tongue, lip, soft palate and posterior pharyngeal wall.

The nodules at first are painless, but within a few days they break down, sloughing, and leave a plug, the removal of which results in a fairly deep crater. They are rather painful in later stages, but heal quite rapidly, new lesions occurring periodically. There is scarring with healing. Regional lymph nodes become tender and somewhat enlarged. Difficulty in eating is experienced as the disease progresses. Recurrence is the rule.

Very little can be done in the way of treatment. Improvement of general health is essential. Cod liver oil, supplementing nourishing food, is recommended. Arsenic, internally, has been found beneficial, and Ormsby suggests 10 or 15 per cent argyrol locally and salicylic acid internally.

Fordyce's disease, an hypertrophy of the sebaceous glands, appears in the mucous membrane of the lips and cheeks in small plaques, pinhead in size, and yellowish-white in color. In some cases they are papular and on palpation appear granular. Occasionally Fordyce's disease is found also in the regions of the alveolar processes, particularly about the molar teeth and the outer surfaces of the lips.

The lesions, consisting of sabaceous glands, usually are symptomless with no pain or ulceration. The etiology of the disease is not definitely known.

Attempts at treatment usually are unsuccessful. Cautery has been attempted but usually is unnecessary and unsatisfactory. Assurance of the benign character of the lesions usually is sufficient.

Scrotal or grooved tongue is characterized by deep grooves or furrows which may or may not be symmetrically placed. The condition is congenital, resulting from some prenatal disturbance. Grooved tongue should not be classed with diseases, as it is symptomless and painless, but occasionally the deep crevices lend themselves to infection from food débris which becomes lodged in the grooves. Usually treatment is unnecessary, beyond reassuring the patient that the condition is harmless.

The cause of *geographic tongue* is not known. The disease begins with several small patches which are smooth, red with a white non-elevated border. The spots enlarge, becoming circles, which widen to meet other circles and form geographic outlines. They usually occur on the dorsum or borders of the tongue. The rings, red in the centers, with white borders, disappear and reappear at intervals. Usually there are no symptoms. The lesions must be differentiated from leucoplakia, mucous patches, and, perhaps, tuberculous lesions. Treatment is unsatisfactory, but alkaline mouthwashes and mild astringents have been found beneficial. The prognosis is always good.

Cheilitis exfoliativa is a rare disease, characterized as a chronic inflammatory condition of one or both lips. Crusts and scales appear and slight swelling may be noted. It is thought by Ormsby and others that the disease is related to seborrheic dermatitis. Relation to eczema also is indicated. Burning sensation and some pain are felt, particularly when the crusts are removed. Fissures may be found beneath the crusts, and the reddened area exposed may bleed easily. The disease resists treatment but *x*-ray and radium have been found valuable. Stellwagen suggests a sulfur ointment and resorcin lotion.

Cheilitis glandularis, may involve both lips, but usually affects only the lower lip. Swelling and tenseness are apparent, and mucous glands enlarge. The mucopurulent discharge which dries on the lip apparently comes from the dilated glands. Sutton and Sutton have noted an associated inflammation of the buccal and pharyngeal mucosa. The disease is benign. Potassium iodide and *x*-ray have been found valuable so far as treatment is concerned.

Actinic cheilitis occurs in summer among outdoor workers and involves the lower lip only. It really is chronic "chapping" of the lips, occurring in individuals sensitive to the sun. The chief symptoms are dryness, scaling and some thickening of the lip. Fissures may be present. Some of these patients have been helped by growing mustaches. Others find applications of cold cream helpful. We wish merely to call attention to the fact that cheilitis and stomatitis may result from the use of lipstick or mouthwash.

Tuberculosis of the oral pharynx is rather rare. Primary lesions are very few, most being secondary to tuberculous disease of the upper respiratory tract. There is some question as to the mode of infection. It may occur by direct contact with sputum, by lymphatics, blood stream and by continuity of tissue.

Cade and Spencer describe several pathologic types: miliary tubercles, tuberculous, cold abscess ulcer, fissure, papillar and lupus.

Secondary tuberculous ulcers are the most common lesions. The early ulcers are usually rather superficial with dirty, sloughing fibers. The edges are irregular and may or may not be elevated. There may be an area of surrounding induration, but this is not the usual thing. Later the ulcers may be rather deep and may be filled with dirty granulation tissue. Lesions of the tongue are most frequent. The tip and base are the most frequent sites. The ulcers are not painful at first. Later the adjacent lymph nodes are tender and enlarged.

Impetigo is of streptococcic origin. It is an acute skin disease which may involve the lips and the mouth and which some-

times appears only as an oral lesion. When lips and mouth are affected, it is called impetigenous stomatitis.

Skin lesions reveal at first small superficial tense blebs, which later increase in size and become flaccid. The contents of the blebs, at first a serous exudate, later become purulent. A reddish or erythematous area usually surrounds the pustule. A yellowish crust forms, the periphery of which may reveal epidermic separation. When the crust is removed, a raw surface is left.

The lesions on the cutaneous side of the lip are similar to those just described. Those appearing on the mucosal side become moist desquamations, which, when removed, show underlying erosion.

Frequent washings of affected parts with a lotion consisting of 1 to 2,000 mercurophen, or metaphen solution containing 5 per cent alcohol constitute the treatment suggested by Prinz and Greenbaum.

Branched fissures may appear on the sides and tip of the tongue. These at first have indurated margins which later may break, resulting in a ragged sloughing area.

Care must be taken to distinguish *tuberculous ulcers* from syphilitic lesions. The prognosis is poor, but removal of carious teeth irritants, the use of alkaline mouthwash, and surgical removal of the tongue are mentioned as means for checking the disease.

Certain deficiency diseases such as *scurvy* and *pellagra* predispose the mucous membrane, particularly the gums, to inflammatory reaction. In scurvy, which results from lack of vitamin C, swelling and hemorrhage of the soft tissue may take place. The teeth may be loosened and secondary infection of the soft tissue may occur. According to Spencer and Cade, if the condition persists, there develops a "fungus ulceration which has a bluish-purplish hue, from which is discharged a foul fluid, and the gums become definitely gangrenous." The prognosis is good. Improvement of diet by giving foods rich in vitamin C, and the use of a hot sodium perborate mouthwash are suggested for treatment.

Pellagra is a deficiency disease, caused by a lack of vitamin G. Beefy red acute stomatitis which later becomes chronic, is characteristic of this disease. Erosion may develop on the sides of the tongue. When pellagra attacks the gums they become spongy and red and bleed easily. Teeth loosen, the breath is fetid, and a burning sensation is felt.

Pellagrous dermatitis on the dorsum of the hands is usually present, and is diagnostic. Neurologic symptoms and diarrhea are present in many cases.

Dietary corrections are an aid to treatment. Citrus fruit juices, tomato juice and fresh meat are considered helpful additions to the diets of those suffering from pellagra.

Denture stomatitis or galvanic lesions of the mouth may be caused by irritations from metals used in dentistry. Idiosyncrasies or intolerance toward certain drugs also may result in disturbances of the mucous membranes of the mouth. Lesions, diffuse or localized, may manifest themselves as macules, vesicles, wheals, edema or hemorrhage.

Certain *blood dyscrasias* such as the leucemias very frequently present lesions of the mouth. The teeth are loose, the soft tissue of the gums is pale, edematous and spongy and the tissues bleed easily with the slightest manipulation. There is usually marked secondary infection and the breath is foul. The neighboring glands are usually tender and enlarged. These signs and symptoms suggest blood dyscrasia until proved otherwise by blood studies and a thorough general examination.

Acetanilid, barbital, and antypirin compounds may produce ulcerations, congestions and blebs. The mucous membrane of the lips, cheeks and tip of the tongue often become bluish in color.

The mercurials, and bismuth, may produce a stomatitis marked by excessive

salivation, swelling, and, in severe cases, ulceration with ultimate sloughing and gangrene. Bluish discoloration of gums around the lower incisors and canine teeth occurs commonly with bismuth therapy of syphilis.

Glossodynia, or burning tongue, is seen quite frequently in middle-aged individuals, usually females. Little is known of its cause, although it is sometimes attributed to gastric disturbance, and some consider it a psychopathic neurosis. The pain usually is characterized as a burning sensation, and is fairly steady. It is felt in borders, tip, or anterior portion of the tongue. There are no demonstrable lesions. Treatment is unsatisfactory, but assurance that the disease is harmless is helpful. Fear of cancer is often expressed by patients.

We have endeavored to describe very briefly some of the common oral diseases. It is hoped that this brief paper will serve as an outline and that the appended bibliography will be used for more complete descriptions.

REFERENCES

Spencer, W. G., and Cade, S. Diseases of the Tongue. London, 1931. H. K. Lewis.

Blair, V. P., and Ivy, R. H. Essentials of Oral Surgery. St. Louis, 1925. C. V. Mosby.

Goodby, Sir Kenneth. Diseases of the Gums and Oral Mucous Membrane. 2nd Ed. London, 1925. Humphrey Milford, Oxford University Press.

Mead, S. V. Diseases of the Mouth. St. Louis, 1930. C. V. Mosby.

Sutton, R. L., and Sutton, R. L., Jr. Diseases of the Skin. St. Louis, 1935. C. V. Mosby.

Ormsby, O. S. A Practical Treatise—Diseases of the Skin. 4th Ed. Phila., 1934. Lea & Febiger.

Prinz, H., and Greenbaum, S. S. Diseases of the Mouth and Their Treatment. Phila., 1935. Lea & Febiger.

The heroic days of the "bare-handed" postmortem examination are over. No autopsy should be performed and no organ should be touched without rubber gloves.

From—"The Postmortem Examination" by Sidney Farber (Thomas).

THE USE OF PHYSICAL THERAPY IN DISEASES OF THE EAR, NOSE AND THROAT

J. L. MYERS, M.D.

Associate Professor of Otolaryngology, University of Kansas Medical School

KANSAS CITY, MISSOURI

FOREWORD

INVENTORS are ever creating new instruments which are being recommended to otorhinolaryngologic workers for treating ear, nose and throat diseases. Much propaganda, through salesmen and printed material, has come to the profession, recommending these therapeutic appliances. Often such exaggerated statements are made relative to results obtained through the use of these various generators of physical energy that the physician is bewildered, becomes skeptical and refuses to use any method of physical therapy.

KNOWLEDGE NECESSARY

The medical profession should realize that before prescribing or attempting to use any of the generators which produce energy found in the spectrum of radiation, they should become familiar with the action of that energy on normal tissue and in pathologic conditions, just as they know the action of drugs.

While it is not necessary for a physician to acquaint himself with the entire spectrum of radiation before using any of the generators which produce energy found in various fields, it is worthwhile to learn of the *universe of energy* by which all are surrounded, to become familiar with some of the research work of physicists, inventors and physicians, and to know something about what they have contributed to therapy in making it possible to use these forces in treating diseases. Having acquired this knowledge, he is better able to orient himself in that universe.

Figure 1 is designed to help in visualizing the whole field in one view, as it shows the electromagnetic wave spectrum (spectrum of radiation), some generators which produce energy in various fields, and also some therapeutic uses of this energy.

THE VISIBLE SPECTRUM

It is common knowledge that sunlight is energy from the sun coming to the earth in undulating, wavelike motion; that a ray of sunlight passed through a prism is broken into seven colors called the visible or solar spectrum; that the different colors are due to the number of waves in each; that the unit for measuring distance from the crest of one wave to the crest of the adjoining wave is the Angstrom (A°), which is 1/10,000,000 of 1 mm. in length; that the wave length on the violet side of the visible spectrum measures 3,750 angstroms, and on the red side the wave length is 7,500 angstroms.

THE SPECTRUM OF RADIATION

On studying the figures on the lower part of Figure 1, one observes that in the cosmic ray field the wave length is about 1/1,000 of 1/10,000,000 of 1 mm. in length, and at the opposite end of the spectrum of radiation, in the high frequency field, the distance from the crest of one wave to another is over 621 miles. The shorter the wave length and the more rapidly the waves come the greater the power of penetration. This explains why the gamma ray penetrates deeper than the Roentgen, and why the cosmic ray has more penetrating power than the gamma ray. As one further studies the picture one sees various fields of energy: (1) cosmic ray; (2) gamma ray; (3) Roentgen ray; (4) ultraviolet; (5) visible spectrum; (6) infra-red; and (7) Hertzian waves (ultra short, short, long

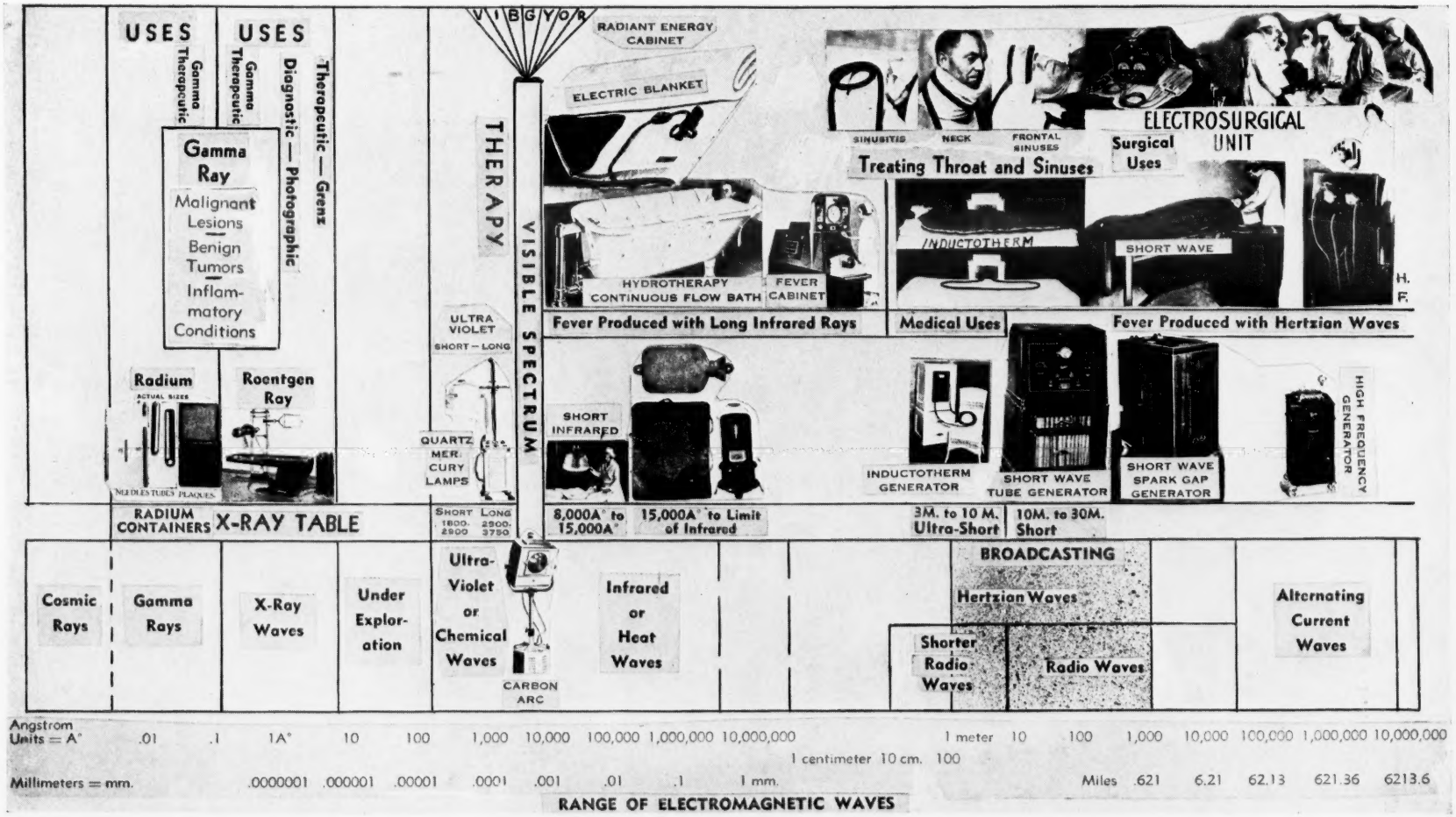

USES
USES
Gamma Therapeutic
Gamma Therapeutic
Diagnostic — Photographic
Therapeutic — Grenz
Gamma Ray
Malignant Lesions
Benign Tumors
Inflammatory Conditions
Radium
Roentgen Ray
NEEDLES TUBES PLAQUES
RADIUM CONTAINERS
X-RAY TABLE
THERAPY
VISIBLE SPECTRUM
V I B G Y O R
RADIANT ENERGY CABINET
ELECTRIC BLANKET
HYDROTHERAPY CONTINUOUS FLOW BATH
FEVER CABINET
ULTRA VIOLET
SHORT — LONG
QUARTZ MERCURY LAMPS
SHORT INFRARED
SINUSITIS
NECK
FRONTAL SINUSES
Treating Throat and Sinuses
ELECTROSURGICAL UNIT
Surgical Uses
INDUCTOTHERM
SHORT WAVE
H. F.
Fever Produced with Long Infrared Rays
Medical Uses
Fever Produced with Hertzian Waves
INDUCTOTHERM GENERATOR
SHORT WAVE TUBE GENERATOR
SHORT WAVE SPARK GAP GENERATOR
HIGH FREQUENCY GENERATOR
Short 1800-2900
Long 2900-3750
8,000A° to 15,000A°
15,000A° to Limit of Infrared
3M. to 10 M. Ultra-Short
10M. to 30M. Short
Cosmic Rays
Gamma Rays
X-Ray Waves
Under Exploration
Ultra-Violet or Chemical Waves
Infrared or Heat Waves
BROADCASTING
Hertzian Waves
Shorter Radio Waves
Radio Waves
Alternating Current Waves
CARBON ARC
Angstrom Units = A°
.01 .1 1A° 10 100 1,000 10,000 100,000 1,000,000 10,000,000
1 meter 10 100 1,000 10,000 100,000 1,000,000 10,000,000
1 centimeter 10 cm. 100
Millimeters = mm.
.0000001 .000001 .00001 .0001 .001 .01 .1 1 mm.
Miles .621 6.21 62.13 621.36 6213.6
RANGE OF ELECTROMAGNETIC WAVES

FIG. 1.

and high frequency)—these form the spectrum of radiation discovered by physicists.

Following the discovery of physicists came inventors who created generators which produce energy of each field (except the cosmic ray) as one observes.

THREE DIVISIONS OF SPECTRUM

In considering the therapeutic uses of this energy one can form three groups:

1. *Waves of Deep Penetration.* The gamma ray, a product of radium, is used for therapy only. The Roentgen ray is used both in diagnosis and in therapy. Since the shortest Roentgen ray is a gamma ray, often the Roentgen machine can be used where radium is indicated. The action of deep penetrating rays is destructive to certain cells.

2. *Waves of Superficial Action.* Ultraviolet, the visible spectrum and the infrared rays are used only in therapy and their action is actinic and thermal.

3. *Waves Generating Heat.* Hertzian waves (ultra-short, short, and long radio waves, and the high frequency), produce heat within and on the body and are used medically and surgically.

TREATMENT OF MALIGNANCIES

A study of medical literature presents evidence that scientific men are constantly giving more attention to the uses of physical therapy in diseases of the ear, nose and throat, because of benefits accruing from the application of these dynamic forces in pathologic conditions.

The increasing number of reports as to the value of this invisible energy in treating malignancies, makes it desirable to call the reader's attention to Figure 1, in order that he may see various generators that are being used today in treating these lesions. Some are proving their worth, others are still in the experimental stage. The list includes: (1) radium—the gamma ray; (2) Roentgen ray generator—the shortest ray is gamma; (3) Hertzian wave generator—the electric knife; (4) Hertzian wave generator—coagulation; (5) high frequency generator—fulguration, desiccation or dehydration; (6) Hertzian wave generator, both the vacuum tube and the sparkgap—diathermy; (7) Hertzian wave generators—fever producing; and (8) long infra-red generators—fever producing.

The successful treatment of malignancies depends on several factors. Hines[1] says that every tumor patient should be "given the benefit of consultation by a group of experts and consultants, inasmuch as no single physician is competent to treat cancer, but the combined efforts of the surgeon, the radiotherapist, the pathologist, the physicist and other specialists are needed to decide upon the diagnosis and treatment in each case."

The securing of a *good* specimen for the pathologist is vital since his report on the histopathologic structure gives the classification of the neoplasm and information as to whether the lesion is sensitive or resistant to radiation.

RADIOSENSITIVITY

Radiologists, in proving that certain cells are sensitive to radiation and thereby are destroyed, made a contribution to conservative treatment of cancer. Cutler[2] on "Radiosensitivity" says: "The outstanding successes in radiation therapy have been accomplished in carcinoma of the skin, lip, tongue, oral mucous membrane, tonsils, pharynx, larynx, etc. It has been noticed that it is precisely in these localities that irradiation has largely replaced surgical procedures." Whether artificial fever makes the tumor more sensitive to irradiation has not been proven, though Warren[3] believes there is efficacy in its use. Fuch[4] advocates using diathermy in treating malignant lesions because of the relief from pain derived thereby.

New and Cabot[5] give encouragement to physicians, and hope to patients in saying:

"1. Malignant tumors of the upper jaw and antrum are curable.

"2. Surgical diathermy and irradiation are the most efficient measures of treatment.

"3. Biopsies should be made in all cases at the time of the operation, as an aid in determining treatment."

RADIATION IN OTHER PATHOLOGIC CONDITIONS

Radiation therapy is not limited to the treatment of malignancies. Robinson[6] reports favorable results in irradiating benign tumors. On the use of the Roentgen ray in inflammatory conditions, Desjardins[7] says, "this method of treatment is not used as widely as it might be, probably because its value is not generally realized." After reading Friedman's report on irradiation in 1,018 consecutive cases of superficial inflammatory disease, one wonders why this therapeutic agent is not used more universally. Hodges and Berger[9] tell of good results obtained in irradiation of "early localized erysipelas in adults, furuncles and furunculosis, granulomas, infected hemangiomas, cellulitis of certain types, rhinophyma, carbuncles, etc." On irradiating cases of bronchiectasis Berck and Harris[10] have concluded "Roentgen therapy in moderately large dosage as the sole method of treatment for chronic secreting bronchiectasis is feasible and successful, resulting in great symptomatic improvement in a considerable proportion of cases."

From the above one can see that a broad field is being covered by research workers in irradiation. As investigators perfect their technique and develop a more accurate method of treatment, we may expect greater progress.

PHOTOTHERAPY

Again referring to the spectrum of radiation, in the range from 1,800 angstroms to the limit of the infra-red field, one finds both visible and invisible light waves. The short ultraviolet rays, which are bactericidal in action, and the long ultraviolet rays, which are biological in action are invisible; the solar spectrum is visible and is both actinic and thermal in action; the infra-red rays (short and long) are invisible and thermal in action.

Laurens[11] says, "There is a general, almost universal, but wrong tendency to limit the biological effects of radiant energy and their therapeutic action to the region between 2,900 and 3,130 angstroms. That radiation is solely or mainly active because the ultraviolet fraction has been unequivocally demonstrated in only a few instances (rickets and infantile tetany). There is considerable evidence on the other hand that the luminous and infra-red rays, even if not specific in their action, aid the ultraviolet rays."

Coblentz[12] says: "Except for the question of intensity and wave length, physically there is no marked distinction between the various regions of the spectrum called 'ultraviolet,' 'visible' and 'infra-red.'"

All of the rays in this portion of the spectrum of radiation are superficial in action. Therapeutically their use in ear, nose and throat work has been exaggerated, and has often proved to be disappointing. However, Krusen[13] offers physical therapists some hope by saying that "even with our present rather limited and chaotic understanding of light therapy, it may be said to be distinctly valuable in a fairly large number of pathologic conditions, and to offer great promise of still further usefulness when more accurate methods of administration are developed, and when we have attained more specific knowledge of therapeutic action of various wave lengths."

HERTZIAN WAVES

Energy of the Hertzian wave field is used both medically and surgically. Medical diathermy—heating through—is used both locally and systemically. The heat is generated by conducting high frequency currents by various generators, through, within or to the body.

By three methods heat is generated within the body:

1. With *conventional diathermy*, the current oscillating from one-half million to

two million cycles per second, metal electrodes are so applied as to conduct the current through the part to be treated.

2. In *short wave diathermy*, we have a high frequency electric field with oscillations from ten million to a hundred million cycles per second. Here the current is conducted into the body by electrodes encased in rubber. These are usually separated from the skin by at least a half inch of felt or towels which prevents the accumulation of perspiration in conducting areas, which might produce burns.

3. With the *electromagnetic induction method*, the current is carried to the part to be treated through a heavily insulated, flexible cable. This cable may be wound around the part to be treated, or it may be wound into a circular form "pancake coil" and laid on the part to be treated. The felt and towels are used here in the same manner, and for the same purpose as previously mentioned.

With conventional diathermy, heat is produced by the current meeting resistance. In electromagnetic induction it is not known how the heat is produced, but it is believed the current passing through the insulated coil sets up within the body "an alternating magnetic flux having the same frequency as the current within the coil," the result being the generation of heat within the tissues.

Research workers are of the opinion that the only therapeutic results obtained in using these generators is through the heat produced, notwithstanding statements to the contrary.

The effects of diathermy on living tissue are: (1) capillaries are opened; (2) blood flow is increased; (3) tissue metabolism is accelerated; (4) rate of exchange between blood and tissue is augmented; (5) oxygen carried into the veins may be increased 33 per cent; and (6) activity of leucocytes in phagocytosis.

To see the effect of heat and cold on blood circulation, the reader should secure from the office of the American Medical Association the movie film on that subject. After seeing that film the reader can better understand the value of diathermy.

In using diathermy in practice one finds it, not to be too optimistic, a helpful therapeutic adjunct when combined with hygiene, diet and drugs. In acute and recurrent sinusitis when drainage has been secured through natural openings or operative procedure, clinically it gives relief from pain and hastens recovery; in acute middle ear inflammations and in mastoiditis after myringotomy and thorough drainage it often proves helpful; in acute laryngitis and tracheitis it may be used advantageously.

Larsell and Fenton[14] say "Diathermy applied for one month's treatment twice weekly to one side of numerous bilateral maxillary sinusitis cases produced rapid clinical amelioration in nearly all; operative removal of tissues disclosed much recent hyperemia with many histiocytes and some fibrous repair, evidently stimulated by the local and enforced temperature elevation."

We find no positive evidence as to the efficacy of short wave diathermy in purulent conditions, as furuncles and abscesses, before drainage is established. Bierman and Schwartzschild[15] say: "It is our definite impression that when drainage does exist, the infected process usually appears to heal very much more rapidly following short wave application than with any other forms of treatment we have used before."

Experience has taught me that intensive short diathermy treatments will not accomplish the results that less intensive more frequent and longer treatments produce.

ELECTROSURGERY

Electrosurgery may be divided into three types, each differing from the others according to the method and current used:

1. Desiccation, fulguration or dehydration. In this method there is a drying out of the tissues by applying the current, monoterminally, from a conventional diathermy generator.

2. Coagulation or cooking of tissue is done by using either the conventional diathermy or the vacuum tube generator and by applying the current from two terminals; the small active terminal is placed in or on a small area to be coagulated, the large, dispersing terminal being placed in contact with the body with a metal plate or a pad.

3. The electric knife is used biterminally—the large dispersing plate is connected to the body, the active terminal is connected to an insulated handle in which is placed a strong, fine needle or very small, thin blade. When the current is turned on the active terminal is applied to the area to be cut and the current from this instrument makes an incision as with a sharp scalpel.

As to the electric generators used in surgery, one needs to know little or nothing, but one must have a knowledge of and be able to adjust the various switches in order to get the proper current for dehydrating, coagulating or cutting.

To learn the operative technique in electrosurgery one should work on dead tissue (beef steak) and on animals until one has become familiar with each modality and knows how to desiccate, coagulate and cut.

FUNDAMENTAL PRINCIPLES

Learning to adjust all switches properly, and acquiring the technique for the use of the different currents surgically does not prepare one to do electrosurgery. The basic and fundamental qualification for the electrosurgeon is a previous experience as an already skilled surgeon. This is gained only through so many years of surgical practice, either alone or under other surgeons, that skill, technique and keen surgical sense are a part of his being. Purchasing surgical equipment never made a surgeon; nor will owning generators which produce the various currents used surgically prepare any man for electrosurgery in general work or any of the specialties.

Team work is necessary in surgery to secure the best results; in electrosurgery it is more imperative. Greater coöperation and more alertness are required of the entire team at the operating table in electro than in any other surgical method, if that is possible.

The ear, nose and throat surgeon will find a place for desiccating in many benign tumors, and, according to Martin,[16] in some malignant lesions. He can use, to his and the patient's advantage, coagulation and the electric knife in malignancies of the neck, face, lips, tongue, pharynx and antrum.

Some men recommend coagulation and some even advocate desiccation as a method for removing tonsils. More harm than good has been done by this propaganda. Personally, I have never seen a case where electrosurgery should replace regular surgery in tonsil work, even though some skilled otolaryngologists maintain there are such. An electrosurgical tonsillectomy is not without pain (postoperative) and not without danger (hemorrhage often occurs); too often much of the tonsil remains in the fossa and the crypts are covered with scar tissue or mucous membrane, thus causing the infection to be thrown back into the system. The most ardent advocate for electrosurgical tonsil work, Dillinger,[17] by his statement condemns the promiscuous use of this method by men not having through surgical training. He says: "This method requires more skill and training than the ordinary surgical tonsillectomy, and no one except a throat specialist should attempt to use diathermy in the throat."

SUMMARY

1. There is dynamic energy in the spectrum of radiation which can be used in ear, nose and throat work.

2. One should be familiar with the action of this energy on normal and pathologic tissue before attempting to use it.

3. In some pathologic conditions more than one generator producing energy in this spectrum may be used advantageously.

4. Before using this energy one should be thoroughly skilled in the line of therapy he practices.

REFERENCES

1. Hines, General Frank T. Government surgeons mobilized to combat increase of disease among ex-soldiers. *Science News Letter*, Sept. 4, 1937.
2. Cutler, Max. Problem of radiosensitivity. *J. A. M. A.*, 103: 1204 (Oct. 20) 1934.
3. Warren, Stanford B. Preliminary studies of effect of artificial fever upon hopeless tumor cases. *Am. J. Roentgenol.*, 33: 75 (Jan.) 1935. Personal communication.
4. Fuchs. Sensitization of neoplasms by means of short waves. *J. A. M. A.*, 106: 2115 (June 13) 1936.
5. New, Gordon B., and Cabot, Clyde M. Curability of malignant tumors of the upper jaw and antrum. *Surg., Gynec., & Obst.*, 60: 971 (May) 1935.
6. Robinson, G. A. Benign lesions of the eye, ear, nose and throat. *Am. J. Roentgenol.*, 33:801 (June) 1935.
7. Desjardins, A. N. Radiotherapy for inflammatory conditions. *J. A. M. A.*, 96: 401 (Feb. 7) 1931.
8. Friedman, A. B. Superficial inflammatory diseases. *Am. J. Surg.*, 25: 107 (July) 1934.
9. Hodges, F. M., and Berger, R. A. Roentgen therapy of some infections. *J. A. M. A.*, 107: 1551 (Nov. 7) 1936.
10. Berck, Maurice, and Harris, William. Roentgen therapy for bronchiectasis. *J. A. M. A.*, 108: 517 (Feb. 13) 1937.
11. Laurens, Henry. Factors influencing choice of source of radiant energy (ultraviolet, luminous and infra-red). *J. A. M. A.*, 103: 1447 (Nov. 10) 1934.
12. Coblentz, W. W. Sources of ultraviolet and infrared radiation used in therapy. Handbook of Physical Therapy, Chicago, 1936. A.M.A. Press, p. 170.
13. Krusen, Frank Hammond. Light Therapy. New York, 1933. Hoeber, p. 163.
14. Larsell, Olaf, and Fenton, Ralph A. Further research on experimental and clinical sinusitis. *Tr. Am. Acad. Otorhinolaryng.*, p. 345, 1934.
15. Coulter, John S. Medical diathermy. Handbook of Physical Therapy, Chicago, 1936. A.M.A. Press, p. 344.
16. Martin, E. G. Cancer, desiccation and chemistry. *J. A. M. A.*, 110: 207 (Jan. 15) 1938.
17. Mock, Harry E. Electrosurgery. Handbook of Physical Therapy, Chicago, 1936. A.M.A. Press, p. 369.

TYPES OF LARYNGEAL OBSTRUCTION AND THEIR TREATMENT

LYMAN RICHARDS, M.D.

Associate Professor of Otolaryngology, Tufts College School of Medicine

BOSTON, MASSACHUSETTS

STANDING as the gateway to the lower respiratory tract, the larynx occupies a commanding position with respect to the normal passage of air into the lungs. An adequate supply of oxygen is a prime requisite for life and any obstructive process interfering with this is at once manifested by a variety of signs and symptoms the appreciation of which is most important for all practicing physicians. In spite of the fact that the larynx is the narrowest portion in the entire respiratory tract, it is nevertheless possible for some degree of obstruction to exist in it without serious embarrassment, but once this tolerated point is passed serious sequelae may develop in an extremely short time. It is the purpose of this paper to consider the major causes of such obstructions and to outline those forms of treatment which in the light of present knowledge have been found to be most successful.

GENERAL CONSIDERATIONS

As in the case of obstruction to any narrow path through which air passes, the localized manifestations of obstruction in the airway, including the lower pharynx, larynx and upper trachea, are associated with the sound of air passing through a narrow orifice—commonly designated as stridor. This sound is most apparent on inspiration and results from the passage of air between the walls of the larynx where, due to various forms of obstruction, it encounters resistance on its way to and from the lower respiratory tract.

This stridor can best be recognized by experience and by previous auditory contact, but exhibits certain characteristic types dependent on the initiating disease. A high pitched crowing tone, associated most commonly with transitory approximation of the vocal cords is seen commonly in the infantile condition of congenital stridor or in the nervous dysfunction of spasmodic croup. Here the vocal cords although temporarily in contact are not essentially abnormal, present no roughened or inflamed area to the air passing through them and hence produce, in this manner, a tone of a relatively smooth quality.

Inflammatory reactions, secretions, membrane or foreign body contained within the lumen of the larynx and attached to or present at various points along its surface, add to the note of stridor a distinctly harsh or roughened quality. The intensity of this inspiratory roughness varies greatly with the extent of the obstruction and the character of the lesion producing it. It may be audible across an entire room or perceptible only on applying the ear to the mouth of the patient. Its early recognition is very important and the physician must be able not only to evaluate any progressive change in its severity but to determine, insofar as means at his disposal will permit, the etiologic factors underlying the obstruction. So long as the various possibilities of the situation are appreciated, so long as the patient is kept under rigid surveillance until the nature and course of the conditions underlying this stridor are known and so long as timely steps are taken to deal with evidences of increasing obstruction, the patient's life will be safeguarded and the practitioner will be discharging a most serious obligation.

Stridor alone, while one of the most important indicative signs of laryngeal obstruction, is but one of a group of symp-

toms which point the way toward its possible existence. Chief among the others is evidence of hyperactivity on the part of the thoracic cage to create negative pressure within the pleural cavity in an effort to compensate for a decreased oxygenation. This is manifested by an exaggerated expansion with distinct indrawing of the softer tissues of the chest at certain points, particularly the supraclavicular spaces, the suprasternal notch and the epigastrium. These chest movements, coupled with an increase in the respiratory rate are at once a most important indication of the difficulty under which the patient is laboring. Such localized retractions of the chest are particularly obvious in infants and children in whom a more flexible wall shows the effect of exaggerated pressure.

The commonly described and sometimes unduly stressed sign of cyanosis, a symptom manifesting inadequate oxygenation of the blood, has for so many years been a traditional sign of high respiratory obstruction, that it has come to be regarded as a classic feature of this condition. In reality, however, severe and urgent obstruction can be present either with absence of cyanosis or more commonly with an ashy gray color of the lips and pallor of the skin which are equally significant and indicative of the urgency of the situation. Cyanosis may appear only terminally when all hope of recovery may well have passed, and should therefore never be awaited as confirmation of the need of immediate relief. Judged by the color of the lips and mucous membrane, adequate oxygenation may be taking place, when in reality a severe form of laryngeal or tracheal obstruction is wearing out a patient's resistance minute by minute.

Almost equally significant and far more common than cyanosis is the symptom of general restlessness on the part of a patient whose intake of oxygen is inadequate. Infants and children particularly tend to exhibit this sign, moving restlessly about, constantly changing position, turning the head from side to side, and waving the arms in indication of general discomfort and insufficient respiratory exchange. Lack of restlessness must, however, never lead to the conclusion that relief of obstruction is not urgent. Young children especially, exhausted by previous efforts to breathe, will eventually give up the struggle and, with increasing cardiac failure, slip into a terminal unconsciousness quietly unless the obstruction is immediately relieved.

Confirming almost any form of respiratory embarrassment is a rise in the pulse rate, but elevations in temperature will depend on the etiologic factor involved, being noted primarily in the infectious diseases and absent in the mechanical forms of obstruction. In the face of these major signs of mild, moderate or severe upper respiratory obstruction, present often in combination or singly, it behooves the practitioner carefully to weigh their significance and, by frequent examination, to form an opinion as to whether the obstruction is increasing or decreasing. Above all, he should never conclude because of the temporarily mild manifestation of the condition, that nothing serious is in the offing, and should avoid the temptation to administer sedatives or hypnotics which, although for the moment apparently alleviating the symptoms and rendering the patient more comfortable, will in reality only mask valuable diagnostic signs.

So far as possible one must determine the cause of the obstruction, whether infectious or mechanical and whether essentially intra- or extralaryngeal. Simulating true intrarespiratory tract obstruction there will frequently be observed the rapid respiratory rate incident to pneumonia, the cyanosis and embarrassment associated with cardiac disease, the striking distress of the patient seized with a bad attack of asthma, and in infants, the puzzling similarity between true laryngeal obstruction and so-called capillary bronchitis. Any of these conditions may produce disturbances of respiration and appropriate forms of physical examination must be

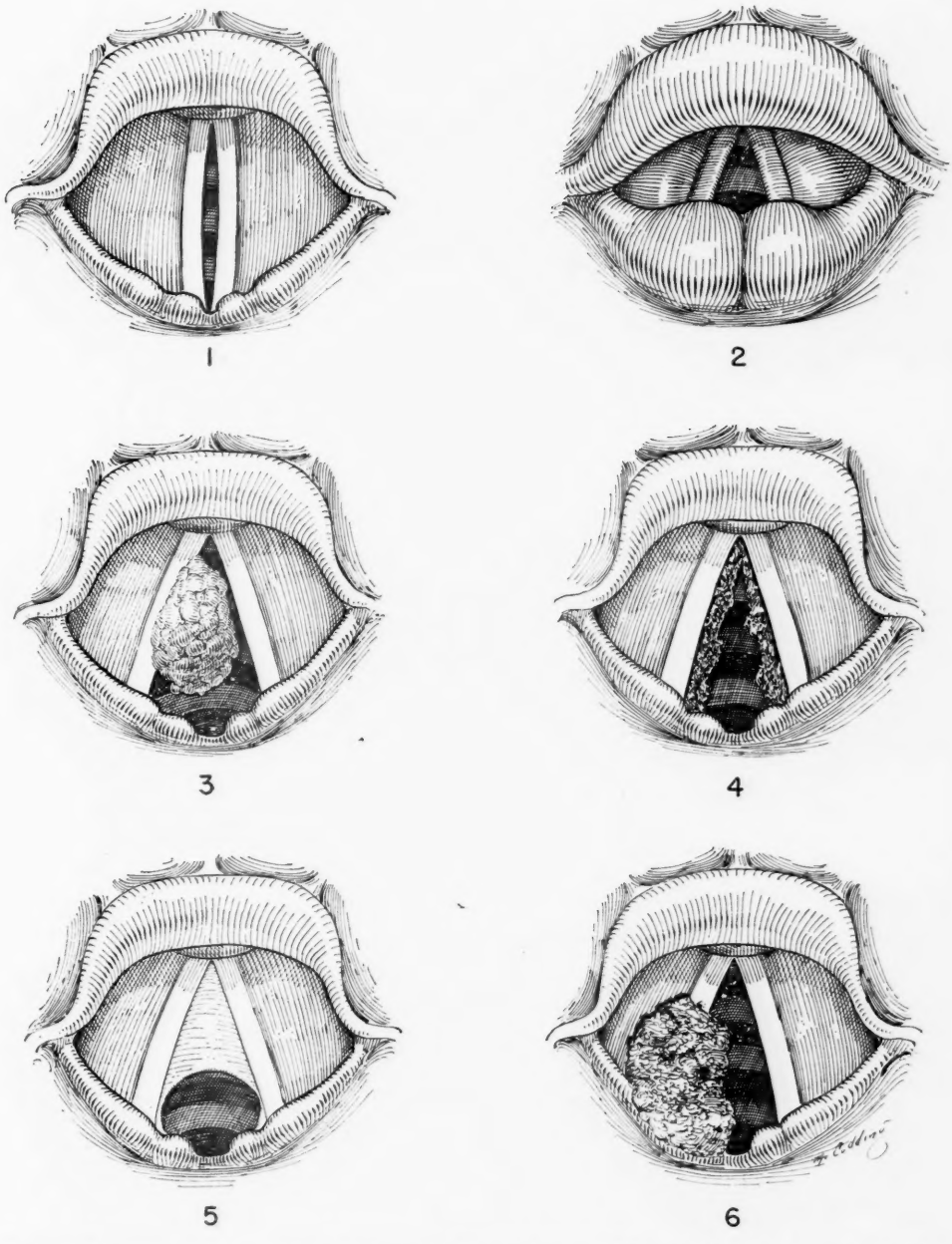


Fig. 1. Representative types of laryngeal obstruction. 1. *Bilateral abductor paralysis.* Obstruction created by close approximation of the cords in the midline, with only a narrow air-space sufficient to sustain life. 2. *Acute streptococcus laryngitis.* Marked edema and hyperemia of the arytenoid and false cords, overhanging the glottis and restricting the airway. 3. *Papilloma.* Benign growth, springing from the cord edges, gradually enlarging until encroaching on the lumen of the larynx. 4. *Acute laryngo-tracheo-bronchitis.* Subglottic accumulations of dry, inspissated secretions along the glottic walls with only the minimum of inflammatory reaction in the arytenoid and false cords. 5. *Chronic cicatricial stenosis.* The result of various inflammatory processes which heal with stenosing scar tissue, here a web in the anterior commissure. 6. *Carcinoma of the larynx.* Malignant disease, advanced to a point at which the growth encroaches on the laryngeal airway.

utilized in order to establish a differential diagnosis. It cannot be too strongly stressed that any patient in whom respiratory obstruction is under diagnostic consideration should if possible be domiciled in a well equipped hospital.

Much can be learned as to the nature of the respiratory obstruction by a careful inquiry into the history of its onset. Relatively rapid developments would suggest an acute inflammatory process while symptoms due to some new growth or slow growing and compressing tumor would arise more gradually. The presence of diphtheria in the visible portion of the throat would be strong presumptive evidence of laryngeal obstruction by this same process. Such obviously diagnostic points as the history of aspiration of a foreign body or even a suggestion of the possibility of such an accident will prove invaluable in locating the cause of laryngeal or tracheal obstruction, provided that the physician will pay heed to what may, on the surface, seem an unlikely suggestion by the patient himself or his family. The presence of fever, an elevation in the white blood count or the respiratory and pulse rate, will all prove helpful in establishing a causal relationship in the face of infectious respiratory obstruction.

Laryngeal obstruction may helpfully be subdivided according to whether the etiologic factor lies outside (extralaryngeal) or inside (intralaryngeal) the larynx.

EXTRALARYNGEAL OBSTRUCTION

Extralaryngeal pathology may by mechanical pressure or inflammatory edema encroach on the laryngeal lumen and in this manner provoke obstruction. The chief location of such pathology is the area, just above the laryngeal orifice, through which air passes from the nasal chambers to the larynx. Inflammatory diseases in this pharyngeal region may at times produce a sufficient amount of swelling to encroach upon the laryngeal airway, with resultant respiratory embarrassment. Such inflammation is often associated with a localized collection of pus, which may not be definitely recognizable or surgically drainable until respiratory obstruction is far advanced. Occasionally acute tonsillitis, when involving already hypertrophied tonsils, will produce such serious encroachment on the pharyngeal airway, as to require immediate tonsillectomy (one of the few situations in which this operation can in any way be called urgent). Peritonsillar abscess, lingual or sublingual abscess, alveolar abscess and cervical adenitis will at times produce such marked swelling in the pharynx as to raise the question of the need of supplementary measures for its relief, entirely apart from treatment of the local condition, and any patient suffering from such pharyngeal inflammation must always be carefully watched for this possibility.

Drainage of localized pus will often suffice to obviate further measures for the relief of this respiratory obstruction. The thickness of the surrounding inflammatory walls of the abscess will diminish and an airway, temporarily obstructed by such edema and swelling, may soon return to normal dimensions. On the other hand, surgical drainage may not be definitely indicated and impending respiratory obstruction must be dealt with without delay, no matter how desirous one may be of avoiding further surgery. Preliminary trial will, of course, have been made of all conservative procedures such as shrinking with astringents, hot irrigations, external hot compresses and steam inhalations, all designed to maintain a maximum patency of the airway in the face of threatened obstruction.

Of particular importance in this connection is the condition of retropharyngeal abscess, commonly seen in infants and children up to the age of 3 years and often unsuspected as a cause of laryngeal obstruction. Its presence should be considered in any case of interference with normal respiratory exchange; a lateral roentgenogram of the neck should be taken to confirm the diagnosis and the utmost

care should be taken in the local examination not to make undue pressure on the tongue with a tongue depressor or mouth gag. Gentle palpation with the index finger will reveal the characteristic elastic fulness of the posterior pharyngeal wall usually to one side of the midline and at times well below the point of visibility through the mouth.

The treatment of laryngeal obstruction incident to retropharyngeal abscess is incision and drainage of the abscess, the technique of which is beyond the scope of this paper. Attempts to deal with the larynx locally or by tracheotomy are ill-advised when drainage of the abscess will suffice entirely to relieve the obstruction.

INTRALARYNGEAL OBSTRUCTION

The causes of intralaryngeal obstruction are such that the limitation in the airway is likely to be more complete and therefore to require more direct measures for its relief than is the case in extralaryngeal obstruction. Since the larynx is anatomically the narrowest portion of the upper respiratory tract it follows that lesser degrees of obstruction produce more serious effects in this region than in portions above or below it. Particularly is this true in children in whom the subglottic space represents not only the narrowest area in the airway, but also the region in which inflammatory lesions are most frequently encountered. Such laryngeal obstruction may be divided into two essential categories, (1) inflammatory and (2) non-inflammatory.

1. *Inflammatory Obstruction.* Any form of acute inflammation of the walls of the larynx can, as a result of swelling of the mucous membrane or submucosal tissues, produce definite glottic obstruction. Outstanding among such inflammatory causes, particularly up until the last five years, was diphtheria. This disease was formerly the cause of death of countless children the world over. It is fortunately becoming increasingly rare as a result, first, of the discovery of diphtheria anti-toxin as a therapeutic measure and more particularly of late of the ability to immunize children sensitive to this disease by the administration of toxin-antitoxin. Unquestionably as time goes on the number of cases of pharyngeal as well as laryngeal diphtheria will become increasingly smaller and one may hopefully look forward in a relatively short time to its complete eradication. Nevertheless, at present, one must still keep diphtheria in mind as a possible cause of laryngeal obstruction.

The suspicion of its existence would be aroused by the discovery of diphtheritic membranes elsewhere in the throat, although laryngeal diphtheria may readily occur without such evidence and cannot therefore be ruled out because of its absence. In the rarer instances in which the patient is a young adult the characteristic appearance of the membrane attached to the vocal cord edges, would be seen in the laryngeal mirror. In children the diagnosis of laryngeal diphtheria can only be properly substantiated by direct laryngoscopic examination of the larynx. This procedure reveals the typical membrane usually attached to the vocal cord and at the same time offers the ideal opportunity for the insertion of an intubation tube. While indirect intubation by the classical O'Dwyer method is still practical, particularly where hospital facilities are not available for direct endoscopy, the latter has so many advantages as to make it greatly to be preferred. More recently there has been some enthusiasm for treating this condition by mechanical removal of the membrane by suction or forceps and foregoing the insertion of a tube unless absolutely necessary. This method obviously necessitates keeping the patient under closest surveillance, until, with the supplementary administration of diphtheria antitoxin, the obstruction subsides.

Acute streptococcic infections of the larynx are responsible for a large number of instances of severe respiratory obstruction, particularly in children. Infection is

often secondary to a similar process higher up the respiratory tract and frequently follows such simple infections as an ordinary head cold or acute tonsillitis. The characteristic pathologic process is an inflammatory swelling of the mucous membrane and edema and hyperemia of the submucosal tissues, particularly of the arytenoid cartilage and the subglottic walls. This swelling and edema are the essential causative agents in the obstructive process and, unlike diphtheria, are rarely accompanied by any definite membrane formation.

Hoarseness, though frequently present, is not so pronounced a feature as in the case of diphtheria since the cord edges are often uninvolved in the inflammatory process. The febrile reaction is apt to be quite high with temperature elevations up to 103° or 104°F., but the outstanding sign is a gradually increasing respiratory obstruction with restlessness and a rise in the pulse and respiratory rate, and, together with this, an intense and ever-increasing general toxicity which becomes in many instances an even more compelling factor in the necessity for alleviation of obstruction than the actual respiratory embarrassment itself.

With such a picture in mind it is at once evident that the administration of antitoxin under the supposition that such a case is of diphtheritic nature is not only futile and misdirected treatment, but entails grave risk to the life of the patient if such obstruction is not at once relieved. There is unfortunately as yet no treatment with the specificity which characterizes diphtheria antitoxin and antistreptococcic serum in general has not proved particularly efficacious in this form of acute laryngeal obstruction. Such a temporary and palliative measure as placement of the patient in a croup or oxygen tent, while possibly effective in mild cases of early acute catarrhal laryngitis, cannot be relied upon for any period of time beyond an early trial and, if ineffectual must at once be abandoned for other and more drastic measures. The drug sulfanilamide may well be worthy of a trial under these circumstances, but the fact must never be overlooked that no medication or form of generalized therapy can ever be relied upon in a case of impending asphyxiation and that when events are progressing unfavorably no procedure can be countenanced except mechanical relief through by-passing of the obstruction at a lower point. There remains, therefore, only the operative procedure of tracheotomy, the details of which will be discussed below. The utilization of intubation under these circumstances, while advocated by some, is in general to be avoided and condemned, because, although effective in temporary relief of the obstruction, it entails subsequent difficulties in extubation and possible residual laryngeal stenosis which militate very strongly against its use.

Another acute obstructive infection involving the larynx is commonly designated as laryngo-tracheo-bronchitis. Sometimes associated with epidemics of influenza, it gives rise within a remarkably short time to a high degree of respiratory obstruction which, although beginning in the larynx itself, tends rapidly to involve the trachea and upper bronchial tree. The pathologic feature is an accumulation of thick, tenacious, almost glue-like secretion along the cord edges and walls of the larynx, particularly in the subglottic region, producing increasing laryngeal obstruction and hoarseness, but differing entirely from the type of true membrane found in diphtheria.

Although post-mortem examinations show this condition to be essentially a streptococcic infection, there is lacking at the outset the intense infiltration and edema of the arytenoids and supraglottic tissues which is seen so commonly in true streptococcic laryngitis. Such cases, however, are marked by the same high degree of toxicity and systemic reaction as in the above described streptococcic infections and the prostration and gradual suffocation of these patients present an

alarming picture which, once seen, can never be forgotten.

All available palliative measures designed to combat this infection have, in the author's experience, proved futile, and tracheotomy, both from the standpoint of relief of obstruction and from that of accessibility for removal of accumulated and inspissated secretions from the trachea and bronchi, is absolutely indispensable.

The above described acute infections of the larynx are, of course, far more common in children than in adults, first because of the frequency of all respiratory infection in childhood and second because the relatively narrower laryngeal passages in the lower age groups permit a lesser degree of inflammatory reaction to provoke a far greater obstruction. One must, however, never forget the possibility of similar laryngeal infections in adults. Their manifestations are more readily observed and diagnosed because of the possibility of direct mirror examination of the larynx, and other warning signs, particularly hoarseness, are likely to appear well in advance of serious obstruction.

2. *Non-Inflammatory Obstruction.* Those conditions arising within the larynx which produce varying degrees of obstruction, but which do not initiate from an infectious process, may be divided, according to their etiology, into three major groups:

(*a*) Some form of spasm or paralysis, arising from a neuromuscular disturbance, may result in a temporary or permanent encroachment on the laryngeal airway.

(*b*) A new growth, either benign or malignant, may restrict the available space within the larynx.

(*c*) The lodgment of some form of foreign body inhaled into the laryngeal box, through its interference with the passage of air, may produce varying degrees of obstruction.

The varied mechanism underlying these three non-infectious processes may create a situation requiring immediate relief if the patient's life is to be saved, whereas, in other instances, the onset of symptoms is so slow as to afford ample time for diagnosis and application of appropriate treatment. The former type is well exemplified by the sudden inspiration of a foreign body into the larynx, the latter by the gradual development of a malignant tumor.

As in other diagnostic problems, differential diagnosis of such obstruction rests first on a detailed history of onset, second on the physical examination and, wherever possible, visualization of the larynx by mirror examination or direct laryngoscopy. Rarely in adults will the dyspnea from laryngeal obstruction be so acute as to preclude this diagnostic mirror examination. In the case of children and infants, the problem is a more difficult one. Certain children, between the ages of 5 and 6, will not infrequently coöperate sufficiently to permit a satisfactory mirror examination of the larynx. With younger children and with older ones who, for one reason or another, will not coöperate, the practitioner is denied the opportunity of this all-important bit of evidence which can only be supplied by resorting to direct laryngoscopy.

LARYNGEAL VERTIGO

Spasm of the laryngeal apperture resulting in transient approximation of the true and false vocal cords may result from any irritant contacting the mucous membrane. This is essentially a protective mechanism, designed to keep intruding substances from gaining access to the lower airways. It is exemplified by the result of water or food being swallowed the "wrong way," by a drop of medication from a syringe striking the unanesthetized mucosa, or by the paroxysmal obstruction seen in whooping cough. In a larynx unduly irritated by vocal abuse the increased irritability of the mucosa may lead to spasm when a drop of nasal secretion strikes it, waking the patient up from a sound sleep. Hoarseness, except in the

presence of chronic irritation will not be present.

The immediate attack can be terminated by raising the arms above the head, holding the nose and slapping the patient on the back. Mirror examination between attacks will quickly reveal the essential lack of true obstructive pathology. The essential element in treatment is the discovery of the inciting factor, reassurance that an attack will not prove fatal, and advice designed to relieve any laryngeal irritation which may be present.

CROUP

A similar form of spasmodic laryngeal obstruction is croup, seen essentially in rachitic, undernourished and neurotic children with flabby laryngeal musculature. Often initiated by mouth breathing and the dropping into the larynx of secretions incident to an upper respiratory infection, it is not in itself an infectious process of the larynx. The onset of croup may be quite unexpected and for this reason is all the more terrifying to the child. For the moment its effect may appear extremely serious. Appropriate treatment, however, will usually result in a cessation of at least the immediate attack, followed perhaps by a similar one within a variable time.

It is important that croup, a self-limited and transitory affection, be not mistaken for some other more serious and prolonged form of laryngeal obstruction in which treatment for croup will not only prove ineffectual but may result in a lost opportunity to relieve the basic cause. As a rule, temperature elevation in true spasmodic croup is slight, the patient's voice during any interim between attacks is usually good and in spite of the apparent severity of the obstruction, the high pitched inspiratory stridor and the difficulty in breathing, there is rarely true cyanosis. A history of previous attacks is a most valuable key to the diagnosis though it is of course no guarantee that because a child has had previous attacks of croup it may not in a given instance be suffering from a far more severe and serious cause of laryngeal obstruction. If appropriate measures designed to relieve croup do not have a reasonably prompt effect, one should be suspicious of some more fundamental condition and steps for meeting this situation should be undertaken without delay. Prevention of subsequent attacks may be achieved by attention to general health, removal of diseased adenoids, inclusion of needed vitamins in the diet and avoidance of cold air in the sleeping room.

Classical methods of treatment dictate the placement of the patient in a warm room filled with steam from a hot water bath, with inhalations through a paper funnel of hot vapor medicated with such a drug as compound tincture of benzoin (one ounce to a pint) contained in a receptacle which can be conveniently kept hot. Ipecac, a time-honored remedy, carried just to the point of inducing vomiting to break up the muscle spasm, is no longer favored by leading specialists. Much can be accomplished by reassurance of both the patient and the parent, and by drawing the tongue forward and inducing regular and relaxed inspiration.

An attack of true croup, alarming as it often seems during its height, is rarely serious, tends to subside under appropriate treatment and leaves no untoward effect behind. Its closest simulator, the inspiratory stridor incident to whooping cough, is usually readily diagnosed by the history of associated cough. The practitioner should ever be on his guard not to make a diagnosis of croup unless the signs and symptoms are present of transitory spasm in a patient otherwise quite well, often subject to previous attacks, and showing no essential hoarseness or cyanosis.

LARYNGEAL PARALYSIS

Laryngeal Obstruction Due to Vocal Cord Paralysis. The neuromuscular mechanism of the larynx is so constituted that the normal glottic opening, triangular in shape,

is maintained by the tension of certain groups of muscles designated as the abductors, which tend to keep the resting larynx sufficiently open to permit ample ingress and egress of air. These abductor muscles are under the control of the recurrent laryngeal nerves, branches of the vagus, which, leaving the parent nerve in the chest, loop around the aorta on the left and the subclavian artery on the right and ascend to reach the larynx.

The triangular appearance of the glottic opening with the cords in approximation anteriorly just behind the thyroid notch and separated posteriorly where they attach to the arytenoid cartilages, is the one commonly seen in a mirror examination of the resting larynx. Interference with this abductor function may occur in such a way that one or both of the vocal cords no longer retain their abducted position during quiet breathing but, pulled toward the midline by the action of the opposing adductor muscles, occupy a new position closely approximating the midline. When this takes place on only one side there occurs a so-called unilateral abductor paralysis of the larynx. This restricts the glottic airway to one half of its former capacity and results in mild respiratory obstruction, never in itself severely embarrassing and noticed only by the patient on unusual efforts of exertion and activity. It requires no treatment so far as its obstructive features are concerned. The voice though weakened is essentially normal since the adductor function is unimpaired. Mirror examination shows the paralyzed cord lying vertically in the midline and the normal cord abducted in the usual position. On a phonatory effort the normal cord not only approximates the paralyzed cord in the midline, but as pointed out by Jackson actually moves or jostles it somewhat beyond the center. This point is of importance in a differentiation between paralysis and the inflammatory fixation of the arytenoid cartilage, in which case there is complete immobility of the affected cord on phonation.

Such a unilateral paralysis may be the result of pathology in the chest or neck involving by pressure or inflammation the recurrent laryngeal nerve on the affected side. Clinical and *x*-ray search should be made for such a condition. Bilateral paralysis with a disturbance in the abductor function on each side creates a much more serious situation, resulting either in urgent respiratory obstruction or, if the cord edges do not completely contact each other, in severe respiratory distress. The condition is most often the result of inflammation, edema, or operative trauma in the neck, such as is occasionally seen as a complication after thyroidectomy. Rarely some chest pathology would be so extensive as to involve both recurrent laryngeal nerves. In addition certain forms of central nervous disease such as tabes and bulbar palsy can also produce disturbances in the function of the recurrent laryngeal nerve, and should be ruled out by complete neurologic and serologic tests.

In the case of inflammatory swelling or operative trauma, the paralysis may come on quite suddenly and create the need for immediate relief of the resultant obstruction. It is obvious that from an etiologic standpoint the condition is far more common in adults than in children. Hence a diagnosis is most readily made by mirror examination of the larynx, which will reveal the vocal cords closely approximating the midline with only a narrow chink between them. This is sufficient for sustaining life, but produces a high degree of stridor which must be relieved in most instances in order to permit the patient to obtain an adequate respiratory exchange. An important diagnostic point under these circumstances is the fact that phonation is still possible and that there is absent the clinical symptom of marked hoarseness which characterizes so many other pathologic conditions of the larynx.

The problem of permanent relief of bilateral abductor paralysis is a complicated one which has taxed the ingenuity of general surgeons and laryngologists for

a long period of time. Numerous operations have been devised with a view to restoration of the airway through either removal of portions of the vocal cord, dissection of underlying tissue or attempts by plastic operation to widen the glottic space in an effort to provide more space. For purposes of immediate relief of urgent obstruction there is only one procedure worthy of a moment's consideration under the circumstances, namely tracheotomy, a detailed description of which will be found on a subsequent page.

It will occasionally be found that obstetrical trauma during difficult delivery will so derange the recurrent laryngeal nerves as to produce a bilateral abductor paralysis. Such a condition might easily be mistaken for an unexplained or cardiac dyspnea (blue baby). However, prompt relief might prove a life saving measure if endoscopic confirmation were available. Rarely such bilateral abductor paralysis will persist into childhood.

A transitory intermittent form of laryngeal obstruction seen in the newborn and in infants is known as congenital laryngeal stridor. It appears in an otherwise perfectly healthy child as a periodic high pitched inspiratory stridor with collapse of the glottic walls due to a congenital weakness in the cartilaginous structure with infolding and indrawing of the epiglottis and arytenoid emminences which tend to approximate each other during inspiration. This stridor appears frequently with crying or exertion and is usually absent during sleep. The condition tends to right itself in due time without any essential treatment, since, as the child grows older, the glottis enlarges and the laryngeal walls become strengthened until they no longer collapse under enforced inspiration. There is no cause for alarm and no likelihood of serious consequences because of this condition so long as its nature is recognized and so long as the picture is not confused with some other fundamental and more serious cause of laryngeal obstruction in a newborn infant or child within this early age group.

A form of this same condition, occurring in nocturnal attacks, known as laryngismus stridulus is described in pediatric literature as a complication of tetany, associated with deficiency of blood calcium and abnormal function of the parathyroid glands.

NEW GROWTHS IN THE LARYNX

New growths springing from the laryngeal walls tend, as they enlarge, to cause increasing respiratory obstruction. In the face of progressive symptoms some form of intralaryngeal pathology should be suspected and confirmed by mirror laryngoscopy. Such growths arise either from the mucous membrane above the cord edges, from the cord edges themselves or from the subglottic walls below the vocal cords. They may be of benign nature, either in adults or in children, or may be malignant, occurring most often between the ages of 30 and 60 years.

When such tumors arise from the vocal cords they will produce hoarseness long before they reach sufficient size to provoke definite obstruction. If, however, they spring from the supraglottic or subglottic region they may reach a considerable extent before they attract attention. For this reason symptoms of even mild laryngeal obstruction merit a careful mirror examination if the advantages of early diagnosis are to be secured. If neglected, obstruction from such growths eventually may be so severe as to require for its relief the same urgent and drastic measures as those mentioned above.

The treatment necessarily varies with the nature of the growth. Papillomata, seen oftenest in children and characterized by repeated recrudescence of the growths, are best removed by repeated and careful excision with blunt cup forceps, great care being taken not to traumatize the adjacent normal mucous membrane. More recently some enthusiasm has been evinced for treatment by repeated exposure to small doses of *x*-ray, but to date this method has had limited and inadequate trial.

The laryngeal surgery involved in dealing with obstructing malignant growths is beyond the scope of this paper.

FOREIGN BODIES OF THE LARYNX

The aspiration into the larynx of some one of the many forms of foreign body is an accident which may at any time befall a child and more rarely an adult. A wide range of objects placed in the mouth can, through the medium of sudden laughter, crying or coughing, be suddenly inspired and if of such a size as to become lodged within the structures of the larynx may create a varying degree of obstruction.

That such an accident has taken place may be immediately recognized by the resultant cough or interference with phonation because of impaction of the foreign body between or near the vocal cords. Moreover, a child old enough to realize what has happened will often report the accident to a parent or older individual. If the obstruction is sufficiently severe, it will at once manifest itself in the characteristic manner, depending on its completeness, with inspiratory stridor, indrawing of the substernal and suprasternal spaces and obvious signs of dyspnea.

There may, however, be no eye witness to the accident itself and a child may be too young to relate to older persons what has transpired. A diagnosis must then be made on the presenting signs and symptoms which vary greatly according to the nature of the foreign body and the degree of obstruction which has been produced. Under these circumstances, it behooves every practitioner to bear in mind that any form of laryngeal pathology, of which the actual cause is unknown, may conceivably be due to foreign body and to leave no stone unturned to determine the correctness of such a suspicion. Time and again either the patient himself or his parents have brought to a physician either a clear story, or at least a definite suspicion, of possible aspiration of a foreign body. Nevertheless, because signs of obstruction in the larynx were inconspicuous and the symptoms consistent with some other, perhaps more common explanation, the possibility of foreign body has been dismissed and the patient allowed to continue to harbor the intruder until some one more shrewd and more willing to lend an attentive ear to the parents' or patient's suspicions has ultimately made the correct diagnosis, often after almost irreparable damage has been suffered through the sojourn of the foreign body within the larynx.

Given the suspicion of such a laryngeal foreign body, an *x*-ray of this region, including the nasopharynx and the chest, should at once be taken in sufficiently varied positions to confirm or exclude this diagnostic possibility so far as it concerns opaque objects. A considerable number of such foreign bodies, particularly toys, coins and pins, are opaque to the ray and will almost invariably be shown in this manner. This will, of course, not be true of the vegetable substances, grasses and a wide range of other foreign bodies. If such objects are definitely lodged within the larynx, signs and symptoms of this condition will usually be present which, if properly interpreted, will at least raise the suspicion of foreign body.

It is surprising how relatively large a foreign body may occupy the confines of the larynx without producing sufficient interference with the airway to demand immediate relief. Coins, open safety pins, discs and a considerable number of flat objects, such as thin pieces of bone, usually enter the larynx and become lodged in the anteroposterior position, leaving thus a relatively large airway available for respiration on either side. Strange as it may seem, even a considerable sojourn of such a foreign body in the larynx does not always result in subsequent edema and swelling of the laryngeal tissues and hence this situation may go on for some time, manifested only by such relatively mild symptoms as hoarseness or coughing, without attracting sufficient attention to make direct observation

of the larynx an obvious requirement for diagnostic purposes. It is in such instances as these that patients have been treated for diphtheria, chronic laryngitis, whooping cough, asthma and a variety of other general conditions, with no suspicion of the possibility of laryngeal foreign body. While it is quite true that these conditions are far commoner causes of symptoms referable to the larynx, nevertheless the importance of retaining foreign body in the back of one's mind as a possible diagnosis until definitely excluded by direct laryngoscopic examination, cannot be overstressed.

So long as the physician maintains this attitude of mind with respect to such cases, particularly in children, there is little likelihood of such mistakes being made. As far as adults are concerned, aspiration of a laryngeal foreign body will almost invariably be recognized at once by the patient and bring him to a physician, under which circumstances direct mirror laryngoscopy will, in most instances, give the true diagnosis. As with children *x*-ray examination will afford helpful confirmation.

The only appropriate treatment of foreign body in the larynx and the only positive means of its diagnosis is direct laryngoscopic examination. Only in this way can the foreign body be brought under direct vision and removed by appropriate operative measures. The latter do not fall within the province of the present article.

Depending entirely on the urgency of the situation a physician may at any time be called upon to relieve laryngeal obstruction without adequate opportunity for study and examination into the etiologic factors involved. While direct laryngoscopic examination should, wherever possible, precede any mechanical by-passing of the obstruction if the most intelligent therapy is to be applied, there will be occasions when lack of facilities or time will preclude it. Under these circumstances there are available two classic procedures, intubation and tracheotomy. The former is less in vogue today than formerly and is considered applicable chiefly to laryngeal diphtheria. The latter has a much more universal application and avoids certain disadvantages which render intubation a less desirable procedure. Discussion of measures for the relief of laryngeal obstruction would, however, not be complete without some account of both intubation and tracheotomy.

INTUBATION

The operation of intubation was first popularized by its inventor O'Dwyer, and was designed primarily for the relief of the obstruction incident to the presence of diphtheritic membranes within the laryngeal lumen, most often in children, for whom it has, on countless occasions, proved a life saving measure.

The apparatus employed is known as an intubation set and consists of a series of hollow tubes, either of hard rubber or metal of graduated sizes and of a shape to conform to the contour of the laryngeal lumen. These tubes are affixed to a so-called carrier, from which they can be dislodged by means of a spring slide when properly placed within the larynx. The original technique can be somewhat simplified by eliminating that portion of the instrument containing the spring and sliding catch, the tube being maintained on the slender obturator by tension on the string attached to the tube and held by the fingers of the intubing hand. A second somewhat similar handle is required for the subsequent removal of the tube when its presence is no longer required within the larynx.

The procedure of introduction of such a tube is as follows: The patient is first securely wrapped in a blanket to prevent the struggling, always to be coped with in any child suffering from respiratory embarrassment. Thus restrained, he is laid on a table with the head held firmly by an assistant. The proper sized tube, selected according to the patient's age

is placed on the introducer, the handle of which is held in the right hand with the physician standing on the patient's right side, facing the head. While the mouth is held open by a gag, the index finger of the left hand is passed over the dorsum of the tongue until the free edge of the epiglottis is palpated. Below and slightly posterior can be felt the two eminences of the arytenoid cartilages. It is into the narrow glottic space between the epiglottis and the arytenoid cartilages, with the vocal cords on either side that the intubation tube is to be introduced. With the left finger as a guide, the intubation tube, held in the introducer, is passed over the back of the tongue just above and behind the epiglottis and gently slipped downwards and forward between the vocal cords until the upper flanged surface of the tube comes to rest against the superior surface of the vocal cords. When the tube is felt to be definitely lodged in the larynx and not, as sometimes happens, to have slipped posterior to the arytenoid eminences and into the esophagus, it is dislodged by slight downward pressure of the left index finger on the flanges of the tube. The carrier is then withdrawn, the string being brought out and anchored to the side of the cheek.

It is one of the dramatic experiences of any physician to note the amazing relief which follows the introduction of such a tube in a child struggling desperately from the respiratory embarrassment incident to laryngeal obstruction. If the tube is properly placed all respiratory effort ceases and the child not infrequently dozes off into a quiet, restful slumber, with easy respiration contrasting markedly with the previous violent efforts incident to the obstruction.

Where such a happy outcome does not immediately follow the introduction of an intubation tube, the possibilities are that by some mischance it has overridden the arytenoid cartilages and slipped into the esophagus, an accident usually due to failure either to make strong traction on the carrier anteriorly or to elevate the handle as the end of the tube is introduced below the glottic rim. More rarely the obstruction may be so far below the larynx, in the trachea, that the tube does not overcome the difficulty.

In certain forms of laryngeal obstruction, particularly those in which the difficulty is due to accumulated crusts, secretions or membranes along the laryngeal walls, the procedure of intubation is ideally designed for relief and avoids certain of the more unpleasant features of tracheotomy. On the other hand, there are certain disadvantages associated with intubation. Among these is, first of all, the necessity of keeping constant and careful watch of an intubated patient against the possibility that the tube will be suddenly coughed up. This accident sometimes arises from the fact that too small a tube was used or from the gradual subsidence of the inflammatory swelling of the walls of the larynx, following the primary relief of the obstruction. Some one capable of replacing the tube must therefore remain at the patient's bedside or within immediate call until further necessity of the tube in the larynx has passed.

Furthermore, certain definite forms of laryngeal obstruction are not ideally suited to this method of treatment. Thus in acute streptococci inflammation, marked swelling of the supraglottic tissues may render the introduction of the intubation tube extremely difficult and even if temporary relief is afforded by its presence its withdrawal is immediately followed by still further swelling with each necessary replacement. Under such circumstances, secondary complications in the form of laryngeal ulceration and chronic stenosis are almost certain to lead to unfortunate consequences.

Finally, the application of the procedure of intubation to laryngeal obstruction due to foreign bodies in the larynx is illogical since attempts to introduce such a tube will only drive the foreign body into a still more inacessible place or into a

position in which it may produce still more complete obstruction.

Hence it is important that the physician have as accurate an idea as possible of the nature of the laryngeal obstruction before introducing an intubation tube and that he consider its use as ideal principally for cases of diphtheritic or membranous obstruction. It is because of the above objections that the procedure of indirect intubation is more and more giving way to the direct method in which the intubation tube is introduced into the larynx only after the latter has been carefully inspected by means of direct laryngoscopy, thus affording an opportunity for accurate diagnosis and the determination of the nature of the obstruction before consideration is given to the introduction of such a tube. Such "visual" intubation can be readily performed, if indicated, as part of the direct laryngoscopic examination.

The length of time during which a tube should be left in the larynx will depend entirely on the nature of the case and the effect of such supplementary treatment as the administration of diphtheria antitoxin. Removal is accomplished by a reversal of the technique of introduction, by insertion of the tip of the extubator into the lumen of the tube. Following extubation close observation of the patient will be required before decision can be reached as to the safety of leaving the tube out. If symptoms of obstruction recur the tube must be reintroduced. If a third introduction proves necessary it is in general better to abandon intubation entirely and to resort to tracheotomy, lest further trauma to the larynx make ultimate extubation impossible and risk the development of ulceration with possible subsequent stenosis.

A procedure somewhat analogous to indirect intubation, much favored by laryngologists during the past few years, is the introduction of what is commonly known as the Mosher life saver. This instrument consists of a long, hollow cannula with a blunt pointed and distally perforated end, curved to fit the general course of the upper airway and designed to be introduced in much the same manner as the O'Dwyer intubation tube. The life saver is carried over the back of the tongue, the epiglottis being identified as in intubation with the left forefinger and the tip of the life saver passed between the vocal cords into the laryngeal lumen.

Once introduced, it can, temporarily, be left in place with relief of the laryngeal obstruction, affording time for further consideration as to proper means for management of the situation in hand. This life saver is extremely useful in an emergency, particularly because of its simplicity; it should never be omitted from the physician's ordinary daily equipment. It is made both in adult and children's sizes. Its use, however is restricted by the same limitations already mentioned in connection with indirect intubation.

TRACHEOTOMY

Rivaling intubation in its often dramatic effect and in many ways superior to it because of its almost universal applicability to all forms of laryngeal obstruction, is the classic procedure known as tracheotomy. Indispensable for those anxious occasions when minutes only are available if the patient's life is to be saved, tracheotomy has been carried out under the most adverse and trying circumstances, when no instrument other than the physician's pen-knife was at hand to do the work. In such a situation, the one and only requisite is an opening into the trachea at a point well below the larynx, made with the maximum speed and with disregard of all other considerations.

For the successful achievement of so-called emergency tracheotomy certain fixed principles of technique must be carried out even in the limited time available. These have been so classically described and repeatedly emphasized by Chevalier Jackson, that one finds it impossible to improve on a summary and repetition of them as laid down by this master laryn-

gologist. Let it be said here that any practitioner who may unexpectedly be faced with such a tracheotomy will do well to have always with him a simple yet adequate armamentarium, carefully set aside, sterilized and packed in a convenient carrier, never to be disturbed except in such an emergency. A satisfactory group of instruments is as follows:

1 sharp scalpel	1 tracheal dilator
1 #1 tracheotomy tube	1 Mosher life saver (2 sizes)
1 #2 tracheotomy tube	Gauze sponge
1 #3 tracheotomy tube	Tracheal tape
12 small curved hemostats	

Such a tracheotomy set should always include the above mentioned life-saver since it is in the execution of tracheotomy that this instrument finds one of its greatest fields of usefulness. Once introduced into the larynx of a patient suffering from severe laryngeal obstruction, it affords a prompt and gratifying improvement in the patient's condition and greatly facilitates the opening of the trachea and introduction of a by-passing tracheotomy tube. The haste sometimes attendant on opening the trachea without such preliminary intubation is thus eliminated together with the increased vascular congestion and resultant bleeding and obscuration of the field of operation. Nevertheless, the opportunity to insert such a life-saver cannot always be relied upon and, for reasons mentioned above in connection with intubation its use is not always advisable. Hence, the practitioner must familiarize himself with the cardinal principles of procedure in executing an emergency tracheotomy without such supplementary aid.

To this end, the patient, restrained, if a child, as rapidly as possible, should lie supine on a table, on an assistant's lap, or even on the physician's own knee, with the head so extended toward the physician's left hand as to bring the trachea as prominently as possible to the anterior surface of the neck. With the knife held in the right hand, the so-called tracheotomic triangle is located by placing the left index finger over the trachea, just as it disappears behind the sternal notch, and the thumb and middle finger just inside the anterior borders of each sternomastoid muscle at the level of the thyroid cartilage. The position of these three fingers outlines thus a triangular area, the apex at the bottom toward the patient's feet, the base toward the patient's head. It is the purpose of the skin incision to bisect this triangle, beginning just below the thyroid notch and extending almost to the suprasternal notch. There should be no hesitancy in making this incision of ample length and one must disregard entirely the temporarily copious bleeding which results from incising the already engorged vessels which lie beneath the skin in this pretracheal region. A single bold and uncompromising incision along this midline should include the skin, superficial fascia and pretracheal muscles down to the anterior tracheal wall. Timid and halfway measures produce only bleeding and avail nothing in the relief of the obstruction. This initial incision must be sufficiently deep to permit the withdrawal of the index finger from its position at the sternal notch for the purpose of palpation, in the depths of the wound, of the tracheal corrugations. With this palpating finger as a guide to the knife, a second incision is made directly through the trachea just below the index finger, which must protect from division the higher cricoid cartilage. So long as this palpating finger identifies the tracheal rings before the second incision is made, no harm can come from false direction and failure of the knife to enter the tracheal lumen. With this second incision into the trachea, dividing two or even three tracheal cartilages, the principal object of the operation has been accomplished.

For the moment, air can be admitted to the trachea by simple insertion into the incision of the handle of the knife (as suggested by Jackson) thus spreading the edges of the tracheal wound, and keeping

it open to permit the unobstructed entrance of air to the lungs. After this important end has been accomplished ample time will be available to stop the active bleeding from the incised superficial vessels and gradually to restore order to a scene which, under the best of circumstances, may be somewhat hectic.

There remains finally the insertion of a tracheotomy tube of proper size for the individual patient and equipped with a cone-shaped obturator which is quickly recovered or blown out by the patient's first active expiration. Tapes attached to the outer edges of the tube and tied behind the neck serve to maintain the tube in position after introduction.

So critical may have been the patient's condition and so severe the degree of obstruction that respiration may not immediately be resumed with opening of the trachea. Let no one be discouraged because of this fact since artificial respiration, begun immediately after tracheotomy, has, on numerous occasions, proved ultimately successful in restoring a life which had apparently been lost. During this process, effort should be made, if possible, to keep blood from running into the tracheotomy tube and definite bleeding vessels should be caught with hemostats, to be ligated at leisure after respiration has again been resumed. So long as a heart beat can be detected, no effort at restoration of respiration should be abandoned, provided an unobstruction airway is afforded to the lung.

There is no gainsaying the fact that such a hasty and at times apparently futile operation violates many of the dictates of modern surgery. It may lack all the aspects of asepsis, hemostasis and desirable surgical accessories but any physician who has dealt with emergency laryngeal obstruction will recognize that otherwise essential considerations must give way to the one and only important matter of the creation of an airway into the trachea, permitting oxygen to reach the lung before death supervenes.

Far more frequently the physician will be confronted with a state of affairs in which there is ample time not only for careful examination of the patient to determine the nature of the laryngeal obstruction, but also for a consideration as to the advisability of tracheotomy, either as a preliminary or as the chief therapeutic procedure. The patient, while not urgently dyspneic, nevertheless presents signs and symptoms which indicate that unless some direct measures can be applied to the cause of the laryngeal obstruction itself, by-passing the latter will prove the most effective means of avoiding the necessity for an emergency tracheotomy at a later time when the situation for executing this may be far less favorable. Thus, in a tracheotomy deliberately planned and tranquilly carried out, time is usually available for transfer of the patient to a hospital where all the facilities for a surgical operation are at hand.

Under these circumstances, the physician is exposed only to the temptation of postponing tracheotomy until the most favorable time for its execution has passed. In spite of the feeling that sooner or later the procedure will be necessary, there is inevitably an inborn desire to hope that perhaps it may be avoided. The physician must first of all make up his mind as definitely as possible as to the intrinsic cause of the laryngeal obstruction and as to whether the condition is in any way likely to resolve spontaneously without surgical intervention. Once convinced that tracheotomy will sooner or later be required, valuable time will only be lost in postponing it and increasing difficulty in its execution will inevitably result.

Particularly in those cases in which the cause of obstruction is an inflammatory lesion in the larynx, associated with edema of the laryngeal tissues, tracheotomy is invariably the procedure of choice. Likewise, in those instances of laryngeal foreign body, in which, for one reason or another, means for its removal are not

readily available, tracheotomy as a preliminary procedure has much to recommend it and the attendant disadvantages are as nothing compared with those which may result from its delay or omission. The insidious but ever increasing obstruction due to some form of new growth, either benign or malignant, such as papilloma or carcinoma, is in general best treated by preliminary tracheotomy.

In recent years numerous authors have stressed the great advantage of the passage of a small bronchoscope into the trachea preliminary to elective tracheotomy. This must, of course, be preceded by direct laryngoscopy which permits inspection of the larynx and so facilitates a correct diagnosis as to the cause of the obstruction. Even if this is of an inflammatory nature there will usually be available sufficient space to pass a small bronchoscope or "life saving" tube into the trachea to be left until tracheotomy is completed. With such a tube in place a free airway is maintained, the patient's struggles are almost entirely eliminated and the operation can proceed with a tranquililty to be secured in no other manner. With the opening of the trachea the tube can be withdrawn and replaced by the customary tracheal cannula.

Properly executed in an operating room with careful attention to anatomic and surgical details, tracheotomy is, all things considered, the most dependable method of relief of impending laryngeal obstruction. The patient should be placed supine on the table, preferably with a sandbag under the shoulders and the head gently but firmly supported by an assistant with the chin directly in the midline. Children will require further restraint by being firmly wrapped in a blanket. The operation should be performed under local anesthesia, since even in cases of mild dyspnea, a general anesthetic is fraught with some risk. Excellent anesthesia in all patients can be secured by infiltration of the skin with 1 per cent novocaine, starting at the sternal notch and carrying a line of wheals up to the thyroid cartilage. Deeper injections can then be made into the subcutaneous tissues, passing slightly laterally to the trachea from below upward, care being taken that the needle is not inserted below the depth of approximately 1 cm. from the surface.

A midline incision in the skin, starting just below the thyroid cartilage, is extended to the suprasternal notch. There is a temptation at first to make this skin incision too short with the result that as one goes deeper toward the trachea, the wound area narrows in a funnel fashion until, when the trachea is finally exposed, an inadequate extent of tracheal wall must be dealt with. Following incision of the skin, the subcutaneous fat and superficial fascia are divided, exposing various small veins which cross the midline. These are readily clamped, ligated and divided before carrying the dissection farther down between the pretracheal muscles, which can be separated by a midline incision and drawn laterally, exposing the pretracheal fascia. If required, further anesthesia can be secured by introducing a small amount of novocaine beneath the latter.

At this point there comes into view the one structure which may in any way cause difficulty, namely, the isthmus of the thyroid gland. This will appear as a smooth, dark red mass lying across the midline, just beneath the pretracheal fascia. In general it is most satisfactory to incise this fascia and then to retract the thyroid isthmus upward. At times, this may lie so low that it is easier to clamp it on either side, cut directly across it and ligate the stumps of the gland thus exposed. In either case, displacement or removal of the isthmus leaves no obstructing tissue between the operator and the anterior tracheal surface. It is highly important that this surface be accurately identified by the palpating finger and that the distinct tracheal rings be felt before incision is made through them.

Ideally, the tracheal wall is best entered through the second, third or fourth tracheal

rings. Above all, it is important to incise the trachea below the cricoid cartilage and the first tracheal ring. It is the so-called high tracheotomy with incision through this upper danger zone which has resulted so frequently in subsequent tracheal stenosis and against which so much has been said by Jackson and other leading laryngologists.

It is often helpful, just before actual incision through the anterior tracheal wall, to insert a hypodermic needle between two of the rings and inject into the tracheal lumen a few drops of 1 per cent cocaine and in this manner greatly to reduce the unavoidable cough incident to the normal irritability of the tracheal mucous membrane. A thin-bladed sharp knife is then used to divide two or three of the exposed tracheal rings and the intervening fibrous tissue between them. Great care should be taken in this incision that the posterior tracheal wall is not traumatized as the knife enters the lumen. At this moment there will be an immediate passage of air through the incision, with slight bleeding from the edges of the wound.

Operators vary in their preference as to the type of tracheal incision, some favoring a simple linear incision with sufficiently wide spreading of the edges of the wound to permit introduction of the tracheotomic cannula. Others prefer to make a crucial incision, the first vertically through the second and third tracheal rings, the second transversely between two adjacent cartilaginous rings. Such a crucial incision immediately affords a somewhat wider opening with greater ease of introduction of the tracheal cannula. Still others, following such an incision, advocate the removal, with some form of ring punch, of the four corners created by the crucial incision, leaving an almost circular opening in the trachea. This is still more suitable for accommodation of the tracheal cannula and has been found by experimental work not to lead to any tendency, after its removal, towards a stenosis or cicatricial obstruction of the tracheal lumen.

With the trachea thus opened, an appropriate sized tracheotomic cannula is introduced. The diameter of the cannula should be only slightly less than that of the tracheal lumen, thus permitting air to pass around the tube and if possible through the larynx. It is usually inadvisable to attempt to close any portion of the skin wound following tracheotomy, barring, possibly, the most extreme edges above and below. Such wounds usually heal promptly and cleanly, but attempts at close suturing entail danger of infection in the deeper portion of the wound. A single folded gauze pad is carried about the tracheal cannula and the latter is held in place by tapes passed through the slotted edges and around the back of the neck. Any accumulation of blood which may have entered the trachea during the procedure, is readily removed by means of suction applied through a flexible catheter introduced into the tracheotomy tube and passed into the lower portion of the trachea.

The normally present cough reflex should not be dulled by the administration of sedatives since on it depends to a great extent the patient's ability to rid himself of subsequent tracheal secretions. To blunt and obtund this reflex only deprives him of his own natural defenses against possible pulmonary complications.

Above all, the physician must realize that from this moment on, the patient is a candidate for the most careful nursing care available and, if possible, should be placed in the hands of a nurse who has at least some fundamental training in such care. The outstanding principle to be followed is the constant maintenance of an unobstructed airway through the tracheotomy tube, and of a toilet and cleanliness of the wound, tube and dressing which avoids any tendency to irritation or infection. In this connection, a factor of prime importance is the care of the so-called inner cannula. This tube, held in

place by a small catch at its upper edge, can readily be removed at any time and should be repeatedly cleansed as often as there is the slightest indication of blood or secretions having become dried within its lumen. This cleansing is carried out by introducing through one end of the inner cannula a bent piece of wire passed through to the other end of the tube, on which is threaded a small folded piece of gauze. This gauze is then drawn back through the entire length of the tube, thus freeing it completely of accumulated blood and secretions. During the first twenty-four or forty-eight hours following tracheotomy, it is often necessary to clean this inner cannula at least every fifteen minutes.

With such a free tubal airway, there should, under normal circumstances, be no audible sound to the patient's breathing, which should be tranquil, comfortable, and, unless there are pulmonary complications already present, unmarked by any essential rise in respiratory rate. Any change in this situation should at once attract attention and should be reported promptly to the physician as indicative of some further obstruction at a point below the tracheotomy tube for which prompt and often specialized treatment may be necessary.

The outer tracheotomy tube should, by preference, be changed once a day, at least during the first week, and this only by the physician himself, aided by good illumination and dilatation of the tracheal wound. The outer cannula is introduced with the obturator in place, well lubricated by an external application of albolene, facilitating the insertion of the tube within the tracheotomic wound. So far as possible, the newly inserted tube should be an absolute duplicate of the original, thus avoiding any change in the contour and irritation from contact of the tube end with the tracheal walls. The avoidance of this trauma will prevent any tendency to bleeding and formation of granulation tissue along the tracheal mucous membrane.

CHRONIC LARYNGEAL STENOSIS

The above described conditions which produce laryngeal obstruction are essentially acute, though varying somewhat in the time before relief becomes urgent. In most instances the larynx was normal before the onset of the disease and with the recovery of the patient after appropriate treatment the larynx returns to an approximately normal state. At times, however, the damages created by certain processes is such that permanent changes take place in the laryngeal structures which are beyond any reparative effort of nature. The cartilaginous framework of the larynx may, as the result of pathologic processes, be so deformed and soft tissues may be so replaced by scar tissue, as to result in serious deformity in the laryngeal architecture. Aside from disturbances in voice production with subsequent hoarseness, these cicatricial contractions may lead to a varying degree of laryngeal stenosis and respiratory obstruction.

Such conditions as perichondritis, fixation of the arytenoid cartilages, tuberculosis and syphilis may all produce changes which even with the complete arrest of the actual disease leave behind a varying degree of restriction of the laryngeal airway. Of a somewhat different etiology but productive of chronic stenosis are a wide range of congenital laryngeal deformities of which the commonest is a web in the anterior commissure. In most instances all these lesions will have required previous tracheotomy, but they differ from the group of cases of acute laryngeal obstruction in that it is impossible, without further attention to the stenosis, to decannulate them. Such stenosis may still leave a residual but greatly restricted laryngeal lumen, or the latter may be so narrowed as to constitute an actual atresia.

Such laryngeal stenosis may, moreover, be the direct result of a tracheotomy for acute laryngeal obstruction in which, through haste or inexpert execution, the

opening was placed too high. As Jackson has so often reiterated, a high tracheotomy, encroaching on the subglottic region or damaging the cricoid cartilage, carries with it grave risk of producing chronic laryngeal stenosis, making it impossible to decannulate the patient and condemning him to a life of prolonged disability unless measures are adopted for restoration of the lumen of the larynx.

The treatment of chronic laryngeal stenosis varies greatly with the etiology and duration of the condition. In those cases in which tracheotomy for acute obstruction is followed by difficulty or impossibility of decannulation because of high tracheotomy, the first procedure is the replacement of the tube. This should be done by a separate tracheal incision, not communicating with the original, lowering the tube to a new location and permitting the former tracheal opening to close. In mild cases this procedure will be sufficient, the subglottic swelling resulting from the malplaced tube will subside and subsequent decannulation will be easily accomplished.

In cases of stenosis due to cicatricial disease in the larynx itself much depends on the etiological factor. In syphilis and tuberculosis systemic treatment is necessary to control the underlying pathology in the body as a whole irrespective of its local manifestations. Once healed the stenosing lesions resulting from traumatic and inflammatory conditions will progress no further and the problem is essentially one of mechanical and surgical ingenuity. Much depends on the age of the patient. In children with chronic laryngeal stenosis there is present the normal factor of growth which tends constantly to aid attempts to restore the laryngeal lumen. In adults in whom this growth factor is no longer present and in whom there is no residual flexibility of the laryngeal cartilages, the difficulties are proportionately greater.

The simplest form of procedure in restoration of the laryngeal lumen in chronic stenosis is endoscopic dilatation, carried out usually from above, sometimes by retrograde methods through the tracheotomic opening. During such dilatation, particularly in children, efforts should be made to induce the patient to use what laryngeal space he has by judicious partial closure of the tracheotomy tube. Jackson has called attention to the fact that such attempts to use the larynx are of great importance in its growth and the restoration of the normal airway.

Dilatation, to be successful, often requires a long period of time and taxes the patience both of the surgeon and the patient. In certain cases the degree of scar tissue as the result of previous trauma or inflammation will be such that simple dilatation will be insufficient to achieve success. Under these circumstances it was formerly customary to resort to more complicated measures such as the operation of laryngoscopy in which, after external opening of the larynx and removal of scar tissue, some complicated dilating apparatus was placed in the larynx in conjunction with special forms of tracheotomy tube. Such operative procedure is at best a tedious and difficult affair, requiring the utmost surgical skill and necessitating prolonged hospitalization of the patient. Detailed description of its execution is beyond the scope of this paper.

More recently Jackson has advocated the somewhat simpler procedure of dilatation of a chronic laryngeal stenosis by the use of rubber core moulds. These moulds, shaped like an intubation tube to conform to the normal laryngeal lumen, are inserted in progressively increasing sizes, and by the steady expansile effect of the rubber, gradually create a new and normally shaped airway. The mechanical problems associated with these methods are by no means simple and involve details excluded by the restrictions of this paper.

SUMMARY AND CONCLUSIONS

1. The cardinal signs of laryngeal obstruction are an increasing respiratory and

pulse rate, stridor, indrawing of the supraclavicular, suprasternal, and epigastric spaces, restlessness and pallor or cyanosis.

2. Acute inflammatory disease, new growths benign or malignant, muscular spasm or paralysis, external pressure, and foreign body account for the majority of cases of acute laryngeal obstruction.

3. Healed inflammatory disease and faulty tracheotomy are the cause of most cases of chronic laryngeal obstruction.

4. Severe acute laryngeal obstruction demands prompt and adequate relief either by intubation or tracheotomy. Chronic stenosis requires either dilatation or plastic reconstruction.

It has been said that the status of a hospital can be judged with accuracy by the character of its department of pathology. . . . Young men seeking internships have been advised to select a hospital largely on the basis of its autopsy percentage.

From—"The Postmortem Examination" by Sidney Farber (Thomas).

THE TREATMENT OF STRICTURE OF THE ESOPHAGUS*

CLYDE A. HEATLY, M.D.

Associate Professor of Surgery, in Charge of Otorhinolaryngology and Bronchoscopy, University of Rochester School of Medicine and Dentistry

ROCHESTER, NEW YORK

STRICTURE of the esophagus is encountered with sufficient frequency in general practice to merit a more thorough understanding of the underlying principles of its management. It must be emphasized from the first, however, that the treatment lends itself to no rigid rules. The selection of procedure must vary with the individual problem as well as with the experience of the physician himself. It might be well to call attention at once to the well known intolerance of the esophagus to all surgical manipulation. Thin walls separate its infected contents from the mediastinal spaces so that blind or overdilatation introduces grave danger of fatal perforation. Only methods of dilatation which utilize recognized safeguards against false passage warrant consideration in this discussion.

Successful management requires first of all an exact diagnosis. For this, careful fluoroscopic and roentgen ray studies as well as direct examination with the esophagoscope are necessary. The importance of the latter is frequently underestimated and treatment is undertaken on the basis of the roentgenologic studies alone. This tendency is to be deplored. An unsuspected foreign body or malignancy might thereby escape detection completely. Cases of malignant changes occurring in areas of cicatricial strictures have been not infrequently observed. The writer has in a few instances discovered malignancy in cases which from the roentgenologic studies alone appeared to be typical instances of cardiospasm. All possible information concerning the location and character of the obstruction should be obtained before any selection of the method of treatment is considered.

With the diagnosis established, certain plans of treatment present themselves. It should be the first aim of the physician, however, to treat the patient himself and not the stricture. Frequently these patients when first seen are badly dehydrated or in an advanced state of malnutrition. In such instances fluids should be forced by proctoclysis, infusion or intravenous methods. This not only improves the condition of the patient, but may actually cause an apparently completely closed stricture to open sufficiently to permit fluids to be taken by mouth and, more important, to permit the swallowing of a twisted silk thread. One of the most common mistakes is to consider immediate gastrostomy as the only method of management under such circumstances. Experience has amply demonstrated that this may actually be avoided in many instances.

As has already been emphasized, the method of treatment of the stricture itself must necessarily vary with the type of case presented as well as with the experience of the physician himself. Strictures of the esophagus fall into four groups: congenital, cicatricial, spasmodic and malignant. Their management will be discussed in this order.

CONGENITAL STRICTURES

Strictures of the esophagus of congenital origin are in all probability more common than is usually supposed. They may be single or multiple and, as in one case reported by the writer,[1] may actually involve a considerable portion of the length of the esophagus. It is important to

* From the Department of Surgery, Division of Otorhinolaryngology, University of Rochester School of Medicine and Dentistry, Rochester, New York.

realize that their existence may not be recognized until adult life. This type of stricture responds far more readily to bouginage than do cicatricial obstructions. Careful dilatations through the full lumen esophagoscope with the Jackson filiform bougies in increasing sizes will result in ultimate cure in most instances. Where gastrostomy has already been performed, retrograde dilatation following the Tucker technique subsequently described is the method of choice and involves no risk providing the fundamental rule of avoiding over-rapid dilatation is consistently observed.

The management of congenital atresia does not fall within the scope of this paper. It might be noted, however, that in these cases gastrostomy alone merely hastens the fatal pneumonia because of the communication which exists between the stomach and the tracheobranchial tree in the majority of instances. Closure of the upper end of the stomach must be combined with gastrostomy as the first step in the management of this condition[2] which thus far has proved universally fatal in its outcome.

CICATRICIAL STRICTURES

It is in this group of cases that we encounter many of the most trying and difficult problems in esophageal stenosis. Chemical escharotics, notably lye, as well as local or systemic infections (especially typhoid, scarlet fever, diphtheria, tuberculosis and lues) are the important etiologic agents. Spontaneous recovery can never be anticipated and untreated cases inevitably progress to marked obstruction or absolute atresia. These strictures may be single but are more often multiple, especially in lye cases where large portions of the esophagus may be replaced by dense cicatricial change. It is important to emphasize that although occasionally annular, such strictures are characteristically eccentric. As the result of stagnation of food and secretions, inflammatory changes as well as frank ulcerations occur which still further increase the hazard of dilatation and, in healing, add to the cicatricial process.

All this must be borne in mind in determining the method of management of these cases. It should be repeated that the first consideration should be the general condition of the patient and not the local problem. Dehydration must be promptly dealt with by all recognized measures and if the stenosis is severe gastrostomy should be promptly performed. Although gastrostomy aids in the treatment by affording a certain means of maintaining nutrition as well as lessening the inflammatory process by putting the esophagus at rest, it must not be hastily undertaken until a thorough study of the esophagus by fluoroscopy, roentgen ray and direct esophagoscopy indicates its complete advisability. It must be emphasized once again that one of the commonest errors is to regard gastrostomy as the indispensible adjunct to all treatment of benign cicatricial stenosis.

The first step in treatment after dehydration is controlled is a thorough direct examination. Inflammatory reaction, if present, should be treated by lavage and the frequent administration of bismuth subnitrate (1 Gm. dry on the tongue every four hours). It is safest in all cases to have the patient swallow a twisted silk thread. Twisted rather than braided silk should be used and silk rather than linen. It is difficult even in normal cases to swallow a linen thread and even braided silk is unsatisfactory. It has been repeatedly demonstrated that a twisted silk thread, slowly swallowed without forcing with large amounts of fluids, will eventually make its way into the intestinal tract through marked cicatricial obstruction even with almost complete symptomatic closure. About 10 or 15 feet of thread should be ingested. Nothing should be tied to the end of the thread to facilitate its passage because this may lodge in the stricture and completely block it. In young children the thread should be introduced through the nose and the hands restrained. The thread should be swallowed slowly,

about a foot per hour, in order to minimize the danger of tangling. When the thread has passed into the intestinal tract it can be pulled sufficiently taut to serve as a safe guide for the passage of dilating sounds, the olive tips of which are threaded over it. By this method many cases of cicatricial stenosis can be successfully managed, as has been so ably demonstrated by Plummer, Vinson[3] and many others. In the presence of a gastrostomy the thread can easily be recovered through the stoma and joined to the upper end to form an "endless" string. This forms the basis for the Tucker method which will be subsequently described.

The thread may also serve as a guide for the introduction of a Levine tube, which, maintained in the esophagus in increasing sizes, has recently been advocated by Hoag[4] as a method of gradual dilatation in benign strictures, particularly those of recent origin. This method, according to Hoag, has the advantage of gradual and constant rather than sudden, intermittent stretching and at the same time reduces the chances of static esophagitis by permitting feeding directly through the tube. The writer has had no personal experience with this method but feels that it may prove decidedly useful in certain cases.

In 1924 Tucker[5] reported his method of retrograde dilatation by means of specially constructed bougies of graduated sizes ranging from 10 to 34 F. Recognizing the multiple, eccentric character of the majority of these strictures, as well as the fact that the esophagus is funnel-shaped below the stricture and thin and dilated (bowel-shaped) above, he reasoned that the safest approach for dilatation, especially in severe cases, was from below. The correctness of this has been attested by the experience of many physicians who have successfully managed large numbers of severe cases without fatality. There are several points to stress in the use of this method. The gastrostomy should be low enough in the left rectus to permit easy access to the esophagus. About two weeks are allowed to elapse before dilatations are begun, in order to permit the gastrostomy wound to heal. Heavy silk or fishline should be drawn through in place of the fine twisted silk thread before dilatations are attempted. In cases where the patient has been unable to swallow the thread, it may be drawn through by a small bougie passed up into the mouth through a retrograde esophagoscope or, as Iglauer[6] has demonstrated, by means of a ureteral catheter introduced into the esophagus by a cystoscope passed through the gastric stoma. The writer has used this method in several instances and can recommend its usefulness. It is of the utmost importance that the size of the bougie should not require force to draw it through the area of stricture and that during subsequent dilatations, the size be increased slowly. Dilatations may be given 2 or 3 times weekly or even daily after 20 F. is reached. The treatments should be continued at semi-weekly intervals at least until the largest size (34 F. in a child and 36 F. in an adult) passes easily. During the time that dilatations are being carried out the gastrostomy tube should be increased in size, keeping it only a few sizes smaller than the bougies employed. This is most important because it prevents great discomfort during the introduction of the bougie through the gastrostomy fistula. Nothing except water should be given by mouth until the 20 F. bougie passes easily. This will greatly reduce the likelihood of static esophagitis.

After the course of dilatations is completed, the gastrostomy fistula should be maintained for at least a year in order to insure against possible recurrence of trouble. A much smaller tube can of course be worn during this time. During this period of observation the esophagoscope should be passed every six to eight weeks in order to detect any possible recurrence. The gastrostomy fistula usually closes spontaneously. Some stricture cases, especially of lye origin, must be kept under observation for long periods. The active period of treatment in such cases may vary

from six months to two or three years. McKinney[7] after an unusually extensive experience said, "Once a lye stricture, always a lye stricture. I do not believe it is ever really cured." One important fact, however, frequently overlooked in connection with strictures in childhood is that the natural growth of the esophagus is a most favorable factor if we can restore an adequate lumen by treatment.

Finally, peroral esophagoscopic bouginage must be mentioned as a method of great value, especially in the presence of a single stricture level. Its use is limited, of course, to those well trained in endoscopic technique. It has the further restriction that it permits inspection only of the upper end of the obstruction which limits its usefulness in cases presenting multiple or severe strictures.

SPASMODIC STRICTURE (CARDIOSPASM; PREVENTRICULOSIS)

The studies of Jackson[8] and Mosher[9] have greatly clarified our conception of this common disorder. It is now recognized that the so-called cardiospasm syndrome actually includes, as Jackson states, "a number or morbid conditions, some functional, others organic, but none of them at the cardia." The obstruction is actually at the diaphragmatic level where the crural fibers on the under surface encircle the esophagus in a pinch-cock like action. Many of these disturbances are of neurogenic origin, resulting in failure of the pinch-cock to open at the approach of the food bolus.

Mosher, however, has demonstrated a large group of cases where stricture, compression from the left lobe of the liver, or marked kinking of the terminal esophagus is the basic cause. Visceroptosis with sagging of the diaphragm is commonly associated with this condition.

In both the functional and organic groups, marked dilatation of the esophagus follows with characteristic chronic inflammatory changes in its mucosa due to stagnating food and secretions. The treatment consists in overdilatation of the diaphragmatic pinch-cock in addition to regulation of diet and esophageal lavage when marked esophagitis is present. While the simple passage of the esophagoscope may cure certain cases, the best results have been obtained by the use of the Plummer hydrostatic dilator or the Mosher barium bougie. These bags are passed under fluoroscopic control. The writer's preference is for the Mosher bag, which is readily visualized under the fluoroscope and easily dilated by air pressure. Great care must be exercised not to overdilate the pinch-cock because of the very real danger of fatal rupture. It has been the policy of the writer not to exceed 5 pounds pressure and to sustain dilatation, if possible, over a period of eight to ten minutes. Such treatment can be repeated at intervals with a minimum risk of rupture. Careful dietary restrictions to avoid rough and irritating foods are usually necessary in these cases. Gastrostomy with retrograde dilatation is seldom indicated. The possibility of reflex causes from abdominal disease must also be considered. The writer has observed cases which failed to respond to all local treatment but cleared completely with the discovery and treatment of an ulcer of the pylorus or chronic gall-bladder disease.

MALIGNANT STRICTURES

The dysphagia produced by malignancy of the esophagus is all too often erroneously considered to be spasmodic in origin. One reason for this is that it is not commonly realized that the dysphagia in such cases is often for a long period intermittent rather than constant in character. The roentgenologic studies are generally diagnostic but in certain instances are misleading or inconclusive. Direct inspection through the esophagoscope with biopsy offers the most positive evidence at our disposal and should be performed in all suspicious cases. The progress of these lesions is to complete obstruction. Whether they should be managed by frequent dilatations through the esophagoscope or by intubation in a

deliberate effort to avoid the unpleasantness of gastrostomy; or subjected to early gastrostomy in order to put the inflamed esophagus at rest and thereby facilitate deep irradiation is a matter which must be individually determined by the experience of the physician as well as by the wishes of his patient. Certainly deep *x*-ray therapy seems preferable to radium in these cases. In the opinion of the writer no absolute rules of management are tenable in this condition which progresses so relentlessly to a fatal termination.

IMPERMEABLE STRICTURE

This serious complication of benign cicatricial stricture is fortunately rare. Heindl and Moersch[10] place its incidence at about 5 per cent. It represents the disastrous result of neglect or improper management. As has already been emphasized, the first rule in the treatment of all esophageal strictures is to maintain a patent lumen. Furthermore, the necessity for frequent examinations in all cases of cicatricial strictures during the first year after dilatations have been completed cannot be emphasized too strongly. When the roentgenographic and direct studies show a severe stricture, it is imperative to attempt to get a twisted silk thread into the stomach at once. The diagnosis of esophageal atresia must be made with care. Cicatricial strictures may close completely for several days and then reopen spontaneously. Again, apparently impermeable strictures may reopen when marked dehydration has been relieved. In certain instances of sudden complete obstruction, direct examination will reveal a foreign body impacted in the stricture rather than a complete closure.

As soon as it can be established by fluoroscopic examination, Roentgen ray, and direct esophagoscopy that complete obstruction is present, gastrostomy should be performed immediately. As usual, it should be so placed as to render the cardiac orifice of the esophagus readily accessible from the gastric stoma.

After several days of rest during which the patient is built up and the wound allowed to heal, the stricture should be studied carefully with regard to its length and possible permeability. Lipiodol is the best contrast medium in this work.

Short strictures without angulation offer the easiest solution. Various procedures have been tried successfully in reported cases. The stricture may be attacked from above by peroral esophagoscopy, from below by the retrograde method or by a combination of the two under expert guidance with the biplane fluoroscope. Peroral esophagoscopy has also been successfully combined with digital guidance and instrumentation from below (Brenneman, Jackson, Wishart and Briggs,[11] Heatly[2]) or from above (Imperatori,[12] Moersch[10]). Iglauer[6] has also reported an impermeable stricture overcome by the introduction of a cystoscope into the stomach and the passage of a ureteral catheter. The fact that the walls above the stricture level are thin as a result of dilatation and infection, as contrasted with the thicker walls and more axial approach from below favors retrograde procedures whenever possible. The management of the impermeable stricture is always hazardous and the greatest care and skill are necessary if fatal mediastinitis from a false passage is to be avoided.

CONCLUSION

The ease with which the esophagus may be perforated must be constantly in the mind of those undertaking the treatment of any type of esophageal stricture. The method employed must vary to some extent with the experience of the physician himself. It is imperative that the general condition of the patient receive primary consideration before local measures are attempted. Careful visual or string guidance, the avoidance of blind, over-rapid or excessive dilatations, as well as conscientious observation for months after the conclusion of active treatment are imperative if successful results are to be accomplished.

REFERENCES

1. Heatly, C. A. Congenital esophageal stenosis. *Arch. Otolaryng.*, 8: 66, 1928.
2. Heatly, C. A. Some problems in esophageal atresia. *Ann. Otol., Rhin. & Laryng.*, 45: 1122, 1936.
3. Vinson, P. The swallowed silk thread as a guide in esophageal instrumentation. *Arch. Otolaryng.*, 13: 94, 1931.
4. Hoag, C. L. Benign strictures of the esophagus: a new method of gradual dilatation. *Ann. Otol., Rhin. & Laryng.*, 46: 327, 1937.
5. Tucker, G. Cicatricial stenosis of the esophagus, with particular reference to treatment by continuous string, retrograde bouginage with the author's bougie. *Ann. Otol., Rhin. & Laryng.*, Dec. 1924.
6. Iglauer, S. Impermeable stricture of the esophagus relieved by retrograde bouginage with the aid of a cystoscope inserted through a gastrostomy. *Tr. Am. Broncb. Soc.*, 1932, p. 51.
7. McKinney, R. Discussion. *Tr. Am. Broncb. Soc.*, 1932, p. 49.
8. Jackson, C. Bronchoscopy and Esophagoscopy. Phila., 1934. W. B. Saunders, p. 347.
9. Mosher, H. P. *Oesophagus. Surg., Gynec. & Obst.*, 60: 403–417 (Feb.) 1935.
10. Moersch, H. J. Impermeable cicatricial stricture of the esophagus treated by a modification of the Iglauer technique. *Ann. Otol., Rhin. & Laryng.*, 44: 407, 1935.
11. Wishart, J. N., and Briggs, W. A case of complete cicatricial stricture of the esophagus. *J. Laryng. & Otol.*, 47: 464, 1932.
12. Imperatori, C. J. Complete atresia of the esophagus. *Tr. Am. Broncb. Soc.*, 1931, p. 74.

Accidental autopsy infections are usually caused by careless technique on the part of the operator, the use of torn or unclean gloves, or the employment of knives and scissors with sharp points instead of rounded-off tips. From—"The Postmortem Examination" by Sidney Farber (Thomas).

THE SURGICAL INDICATIONS IN PERFORATION OF THE ESOPHAGUS BY FOREIGN BODIES*

JEROME R. HEAD, A.M., M.D.

Attending Surgeon, Cook County and Hines Hospitals

CHICAGO, ILLINOIS

PERFORATION of the wall of the esophagus is an extremely threatening accident. Leakage of infected contents and air into the periesophageal tissues in the neck or thorax leads to infection and may result in a rapidly fatal cellulitis, mediastinitis, empyema or pericarditis. Mediastinal abscess may develop and occasionally there is fatal hemorrhage from injury to the aorta or carotid. Jackson says that mediastinal emphysema may cause death without a complicating infection. Perforation into the trachea or bronchus leads usually to fatal pneumonia, abscess or gangrene. When one is confronted with the accident it is important to make rapid and drastic decisions.

It is the purpose of this paper to present five cases which I have observed over the past few years and from a review of the literature summarize accumulated experience and current opinion as to the surgical indications.

REPORT OF CASES

CASE 1. Dr. O. Y., a dentist 37 years old, arrived late at a dinner party and ate chicken à la king hurriedly. A piece of it became lodged in his throat and thereafter he had increasing pain and dysphagia. He entered the hospital the third day after the accident. He was weak and prostrated, was unable to swallow liquids and had severe pain in the lower neck. His temperature was 103°F. and his white blood count 16,000. The neck was diffusely swollen, but there was no crepitation.

Fluoroscopy performed during the ingestion of a small amount of barium mixture revealed obstruction of the esophagus at the level of the sixth cervical vertebra.

Through the esophagoscope a three-cornered piece of chicken breastbone was removed from this level. Several c.c. of pus was evacuated from a perforation in the right posterior esophageal wall.

The following day the fever was still high and some cyanosis and difficulty in breathing had developed. Examination of the chest revealed bilateral paravertebral dulness posteriorly and numerous coarse bronchial râles in both lungs.

An *x*-ray taken the third postoperative day showed a marked widening of the entire mediastinum. Physical examination revealed dulness and a friction rub at the left base posteriorly.

The next morning a right cervical superior mediastinotomy was performed and a small pocket containing about 5 c.c. of dark watery fluid was entered. The wound was packed with iodoform gauze. Following the operation the patient continued to have increasing difficulty in respiration and died on the second postoperative day.

Autopsy revealed two perforations of the esophagus, one in the right and one in the left posterior wall about 2.5 cm. below the pharynx. A 3 mm. probe passed readily through the perforation on the right into a sinus which crossed from right to left in the mediastinum anterior to the esophagus and widened out at the level of the bifurcation of the trachea to a maximum circumference of 4.5 cm. At this level it lay between the arch of the aorta on the left, the esophagus on the right and the base of the heart anteriorly. The sinus then passed downward to the diaphragm, dissecting the aorta from the pericardial sac and perforating into the left thoracic cavity just above the diaphragm.

Acute pleuritis and pericarditis were the causes of death. The mediastinal infection was limited to the well walled-off sinus.

In this case perforation and infection had occurred before the patient presented himself for treatment. While it is probable that the

* Read before the Chicago Surgical Society, December 10, 1937.

train of events which led eventually to death had already been set in motion, it is certain that the chances for recovery would have been much greater had the site of the perforation been exposed and packed on the day of admission.

Case II. Mrs. L. P., a housewife 60 years old, entered the hospital for dilatation of a spastic cardia. Esophagoscopy was performed first to rule out the possibility of neoplasm. At the termination of this procedure the patient developed severe dyspnea, and marked substernal and upper dorsal pain aggravated by swallowing. Examination revealed emphysema above the right clavicle and absent breath sounds over the right lung. An *x*-ray disclosed a right pneumothorax. The patient improved gradually during the day and although her temperature was 101°F. that evening, both fever and symptoms decreased on each succeeding day. No fluid formed in the pleural cavity and the air was gradually absorbed.

In this case perforation of the esophagus into the right pleural cavity healed without the development of serious infection. Operation was withheld because the patient's condition improved rapidly and because interference would have had to be radical and dangerous—a thoracic mediastinotomy.

Case III. Miss A. H., a school teacher 45 years old, entered the hospital at 6:00 P.M. For luncheon on the same day she had eaten chicken à la king, had felt something lodge in her throat and thereafter had had increasing dysphagia and submanubrial and upper dorsal pain. Her temperature was 97 degrees. Physical examination was not done.

Esophagoscopy performed two hours later revealed an obstructing mass at the level of the bifurcation of the trachea. Some bleeding was encountered and it was impossible to say whether the obstruction was caused by a foreign body or a tumor. Nothing was removed. Immediately after the esophagoscopy she complained of a great increase in thoracic pain and became dyspneic and cyanotic. Emphysema developed in the neck and over the upper thorax.

I saw this patient six hours after the esophagoscopy and twelve hours after the lodgment of the foreign body. Respiration was difficult and grunting, the skin was cyanotic and the emphysematous swelling of the neck marked. Examination of the chest revealed tympany and absence of breath sounds on the right. No *x*-ray was taken. A needle was inserted in the right midaxillary line and a large amount of air and thin sanguineous pus was evacuated and this brought immediate and marked relief. To combat the pleural infection and relieve the pneumothorax and emphysema, a catheter was inserted into the pleural cavity and constant suction applied. Difficulty in swallowing was intense and there was a constant overflow from the esophagus into the pharynx with considerable aspiration of secretion. An *x*-ray taken the next day showed that the lung was completely expanded and that there was no air or fluid in the pleural cavity. The patient grew steadily worse, however, and died the same day.

The autopsy revealed a three-cornered piece of chicken bone with considerable meat attached lodged in the esophagus just above the bifurcation of the trachea. On the ventral wall of the esophagus there was a necrotic area with perforation. Suppurative mediastinitis and periesophagitis were present. There was a well drained acute empyema in the right pleural cavity, and both lungs were filled with frothy bloody fluid.

This must probably be considered an instance of instrumental perforation, although it is difficult to believe that pus could have formed in the pleural cavity in the six hour interval between esophagoscopy and aspiration. It was hoped that catheter drainage and suction of the pleural cavity would drain the esophagus and mediastinum as well. The patient would probably have had a better chance to survive if we had been more radical and had done a transpleural esophagotomy, removed the foreign body, repaired the esophagus and left suction tubes at the site.

Case IV. Mr. B., 65 years old, while eating stewed lamb, felt something lodge in his throat. He had a moderate amount of dysphagia with submanubrial and upper dorsal pain on swallowing, and consulted a physician the next day. Fluoroscopy and *x*-rays showed partial obstruction of the esophagus at the level of the suprasternal notch. There was no fever. Esophagoscopy was performed at 11:00 P.M. the same day, twenty-nine hours after the accident, and a small piece of bone removed from a point 22 cm. from the incisor teeth.

During the night the patient had increased deep upper thoracic pain on swallowing and

at 7 A.M. had a chill. At this time his temperature was 102 degrees.

I saw him three hours later. Physical and *x*-ray examination revealed a normal chest and mediastinum. There was deep tenderness in both supraclavicular spaces, but no evidence of emphysema. He was transported by ambulance to the Evanston Hospital and a cervical mediastinotomy was planned. When he reached the hospital at about 3 P.M., his temperature had fallen to 99.4, and his symptoms had markedly decreased. Dr. Karl Meyer saw him in consultation, and in view of the improvement in his condition, we deferred operation. Postural drainage and intravenous and rectal feedings were inaugurated. With decreasing evening elevations his temperature returned to normal over the course of a week. Fluids by mouth were then started and he was discharged from the hospital on the tenth day.

This case requires no comment further than to say that if one is to operate on the cases that need operation he will necessarily operate on many who might have recovered spontaneously.

Case. v. Mr. M. M., a law student 23 years old, felt something lodge in his throat while eating turkey wings for his evening meal. He continued to have dysphagia and pain low in the neck and consequently came directly to the hospital. Physical examination showed no swelling or tenderness of the neck. The temperature was normal. An *x*-ray revealed a small piece of bone in the esophagus at the upper thoracic aperture. Esophagoscopy was performed immediately but the bone was not visualized.

The next day he experienced increased pain and difficulty in swallowing and his temperature rose to 101°F. Examination revealed emphysema in the suprasternal notch and deep tenderness in the neck, greater on the left side. An *x*-ray showed that the bone was still present and verified the presence of air in the periesophageal tissues.

Left superior mediastinotomy was performed that evening, twenty-four hours after the onset. No obvious infection was encountered. Although the foreign body could be palpated, an esophagotomy was not performed. The mediastinum was packed off with iodoform gauze.

The patient improved gradually and the return of ability to swallow soon proved that the foreign body had been passed. The temperature became normal on the fourth postoperative day. He was discharged well two weeks after the onset.

REVIEW OF THE LITERATURE

The esophagoscope was developed by Kussmaul, Störck and von Mikulicz during the latter decades of the nineteenth century. Prior to this work, the lodgment of a foreign body in the esophagus was an extremely serious accident which carried a high mortality. Attempts were first made to force it downward with a sound or bougie or to grasp it blindly with an esophageal forceps or to extract it by means of a coin catcher or probang. Instrumental perforations were extremely common and not infrequently the patient died of mediastinitis either before or after the foreign body had been removed. If blind methods of dislodgment were unsuccessful, resort was made to external esophagotomy, if the article was in the upper esophagus, or to gastrotomy if it was located in the lower portion. Kaloperopulos collected 217 cases of esophagotomy, in which the mortality was 20.93 per cent. Balacescu and Cohn collected 323, with a mortality of 26.5 per cent. In the cases which they had operated upon it was only 12.5 per cent. These statistics are of current interest because when esophagoscopy is unsuccessful the operation is still occasionally indicated.

Table I

SUMMARY OF SERIES OF CASES OF FOREIGN BODY IN THE ESOPHAGUS REPORTED IN THE LITERATURE

Author	No. of Cases	Removed by Esophagoscopy	Deaths	
			No.	Percentage
Jackson	204	...	4	1.9
Starck	78	73	1	1.4
Schlemmer	529	508	15	2.8
Kahler	225	...	8	3.5
Vinson	135	...	6	3.5
von Hacker	78	...		

The development and perfection of esophagoscopy made a radical change in this rather gloomy picture. As early as 1909 Starck reported seventy-eight cases of foreign body in the esophagus, in seventy-three of which the foreign body was removed by the esophagoscope, with no accidents and no mortality. In the five cases in which esophagoscopy was unsuccessful and esophagotomy was performed, one patient died, a mortality of 20 per cent for the operation and 1.4 per cent for the whole group. Among 529 cases of foreign body reported by Schlemmer in 1920 from Chiari's clinic in Vienna, in 508 the foreign body was removed via the esophagoscope. There were fifteen deaths in the whole group, a mortality of 2.8 per cent. It is interesting that Schlemmer stated that a foreign body was found in only one out of three cases in which esophagoscopy was performed for suggestive symptoms. Kahler reported 225 cases, in twelve of which there was perforation and in eight of which the patient died, a mortality of 3.5 per cent. Vinson recorded 135 cases, with six deaths, a mortality of 4.4 per cent.

In spite of this great improvement in results, foreign bodies in the esophagus still occasionally cause trouble. Perforation and periesophageal infection may occur before the patient comes to esophagoscopy or may result from the instrumentation. McGibbon and Mather have emphasized the fact that a sharp foreign body can penetrate the esophagus while passing through it and that the whole train of sequelae may result without actual impaction.

When perforation occurs it is important to know what the train of events is likely to be and what procedure is indicated to prevent or cure the conditions which may ensue.

To understand what may occur it is important to recall the relations of the esophagus in the neck and mediastinum. In the neck it lies behind and slightly to the left of the larynx and trachea and in close relationship to the common carotid artery. Throughout the thorax it is covered on each side by the mediastinal pleura. In the upper part it lies behind the trachea; below the bifurcation it runs behind the heart and pericardium and in close relationship to the descending aorta.

In the neck perforation may be into the periesophageal tissues or into the trachea or the carotid, innominate or subclavian arteries. Infection and edema or abscess formation may cause laryngeal or tracheal obstruction, a complication not uncommon in children. The event chiefly to be feared is, of course, descent of the infection into the mediastinum. The anatomy of the cervical and mediastinal spaces and the routes by which infection travels from the neck to the thorax have recently been well worked out by Lambert and Berry and by Furstenberg. It is important to remember that although infections in other parts of the neck rarely descend into the mediastinum, those occurring in the periesophageal spaces do so with extreme ease and frequency.

Perforation of the thoracic esophagus may be into the mediastinal space or into the trachea, pleura, lung, aorta, pericardium or heart. The perforation may be direct or by infection and erosion.

Fatal hemorrhage from perforation of the aorta, innominate or carotid is common. Ter-Oganesjan collected fifty cases and reported five of his own. Killian reported forty instances in his series of 360 operated cases, in some of which the hemorrhage was spontaneous, but in many it was from postoperative erosion of the carotid, a common complication of esophagotomy and cervical mediastinotomy. Clerf reported a case in which the point of a safety pin penetrated the wall of the left ventricle. In Kahler's series of twelve cases of esophageal perforation two patients died of hemorrhage. Many single cases have been reported and practically every series contains additional instances.

The infections which follow perforation of the esophagus can be classified as follows:

1. Periesophagitis—a mild cellulitis which subsides without extension or localization
2. Localized periesophageal cervical abscess:
   (a) Small, draining into the esophagus
   (b) Large, encapsulated
3. Mediastinitis
   (a) Acute, spreading
      (1) As extension from cervical cellulitis
      (2) As result of local perforation
   (b) Localized abscess
      (1) As extension from cervical cellulitis
      (2) As result of local perforation

1. *Periesophagitis.* If perforation of the esophagus led always to spreading infection or abscess formation, the surgical indications would be relatively simple. Many patients in whom emphysema, swelling, pain and fever are present, giving certain evidence of perforation, recover in the course of a few days without surgical interference. Others in whom definite perforations are observed at esophagoscopy develop no signs or symptoms of infection.

2. *Localized Periesophageal Cervical Abscess.* The only evidence that a small abscess is present may be pus seen exuding from a perforation after removal of the foreign body. Drainage into the esophagus being sufficient, this subsides spontaneously.

Localized large cervical abscesses which produce marked signs and symptoms and require surgical drainage are common. Localization is most apt to occur if the perforation has been in the pyriform sinus. Large abscesses may rupture into the esophagus or into the trachea, in the latter case producing pulmonary gangrene and abscess. They may also cause septic thrombosis of the jugular vein with secondary septic infarcts of the lungs. Drainage may be followed by fatal hemorrhage from erosion of the carotid artery. Pressure on the larynx and trachea may cause serious interference with respiration.

3. *Mediastinitis.* Mediastinal infection, whether it descends from the neck, as it so often does, or follows perforation of the thoracic esophagus, may form a localized abscess. These frequently point above the clavicle. They may rupture into the esophagus or trachea or into the pleura or lung. Rupture into the trachea is almost invariably fatal. Erosion and osteomyelitis of the vertebrae have been frequently reported.

More often the infection is acute, spreading and unlocalized, and it may cause death in from twenty-four to forty-eight hours. In nearly every instance the infection extends to one or both pleural cavities and often to the pericardium. It is important to remember that empyema and pericarditis are usually accompaniments of mediastinitis, while an opening may be made directly into the pleural cavity with the production of spontaneous pneumothorax or empyema. Killian reported a case of perforation through adhesions directly into the lung.

Mosher and Kramer have reported instances of septic thrombosis of the azygos vein.

SYMPTOMS AND FINDINGS

The symptoms of esophageal perforation vary with the level and the complications. Increasing painful deglutition with fever, emphysema, and swelling of the neck is the characteristic picture. Emphysema may be absent. Dyspnea and cyanosis from pressure on the larynx and trachea are common, and there is often marked overflow of secretions into the pharynx and mouth. The pain is usually in the lower cervical and submanubrial region, extending through into the back. Low perforation may give few symptoms or signs.

In mediastinal cellulitis and emphysema, dyspnea, from compression of the trachea, and cyanosis, from engorgement of the cervical veins, are the result of increasing pressure. Jackson said that perforation of

the esophagus may give rise to sufficient mediastinal emphysema to cause death without the development of infection, and the occasional very rapid progression to death in cases of acute mediastinitis does speak for a mechanical rather than a toxic process. The dyspnea and venous engorgement point in the same direction. It is difficult to see how any infection can cause death in such a short time, and in reviewing the literature, one comes to the conclusion that only rarely is the mediastinal infection of first importance. It seems probable that the early deaths are caused by an increase in the mediastinal pressure and the late ones by empyema and pericarditis.

The *x*-ray examination is of considerable importance. It will, of course, reveal the nature and exact location of opaque foreign bodies. The presence and location of non-opaque objects may be determined by fluoroscopy after ingestion of opaque substances or capsules. Minnigerode and Iglauer have called attention to the fact that perforation is often evidenced by the presence of air in the periesophageal tissues. Perforation in the neck usually causes an increase in the distance between the spine and the trachea. Abscesses in the mediastinum cause displacement of the esophagus, and acute mediastinitis produces a characteristic *x*-ray picture—namely, a fuzzy-edged widening of the mediastinal shadow.

ANALYSIS OF STATISTICS

As a basis for formulation of a comprehensive picture of the condition seventy-two cases of esophageal perforation have been collected from the literature. To avoid assembling a group of unusual manifestations only those cases have been included which have been reported in series as representing the complete experience of an author or clinic. The seventy-two cases were taken from the reports of the following writers: Alpin, Carmody, Clerf, Jackson, Kahler, King, Kramer, Lederer and Fishman, Lynah, McGibbon and Mather, Miller, Myerson, Orton, Schlemmer, Sheehan, Starck, Wright, and Yankauer.

The nature of the foreign bodies which caused the perforations is presented in Table II. It is important to note that most of these are sharp pointed objects and that the incidence of the different types varies widely from that of all foreign bodies of the esophagus. In Killian's series false teeth comprised 45 per cent, and in children coins were the most common. That teeth caused perforation in but one case in this series and coins in but four indicates that these foreign bodies rarely cause perforation and are therefore far less dangerous than bones, pins and other pointed objects.

TABLE II
TYPE OF FOREIGN BODY IN SEVENTY-TWO CASES OF ESOPHAGEAL PERFORATION REPORTED IN THE LITERATURE

Foreign Body	No. of Cases	Percentage
Bones	38	54.2
Chicken bone	14	
Meat bone	17	
Fish bone	7	
Safety pin	8	11.4
Coin	4	5.7
Collar button	2	2.8
Jackstone	2	2.8
Toothpick	2	2.8
Piece of brass	1	1.4
Thumbtack	1	1.4
Needle	1	1.4
Rivet	1	1.4
Glass	1	1.4
Teeth	1	1.4
Not mentioned	8	11.4

The location of the foreign bodies which caused perforation is shown in Table III.

TABLE III
LOCATION OF FOREIGN BODY*

Region	No. of Cases	Percentage
Upper cervical	5	7.1
Lower cervical	39	55.5
Upper thoracic	7	10.0
Lower thoracic	5	7.1
Not mentioned	14	20.0

* In two cases not listed here, perforation was caused by instrumentation and not by a foreign body.

These figures approximate those of Schlemmer and Killian. The latter stated that approximately 90 per cent of esophageal

foreign bodies are found in the cervical esophagus. Schlemmer, in 525 cases, found the incidence as follows: 72.3 per cent in the upper esophagus and 27.7 per cent below the bifurcation.

The incidence of the different types of complications and the mortality in each group are shown in Table IV. The importance of the fact that twenty-six patients, 36.1 per cent, recovered without the development of serious infection and without surgical interference will be discussed under the heading of treatment.

TABLE IV
TYPE OF LESION

Lesion	No. of Cases	Deaths	
		No.	Percentage
Periesophagitis	19	0	0
Small cervical abscess perforating into esophagus	7	0	0
Large cervical abscess	7	2	29
Acute mediastinitis	13	13	100
Simple mediastinitis (no autopsy)	5		
With empyema	4		
With empyema and pericarditis	3		
With septic thrombosis of the azygos bone	1		
Local mediastinal abscess	11	2	18
Empyema	2	1	50
Pneumothorax	2	1	50
Hemorrhage from perforation of great vessels	5	3	60
Septic thrombosis veins	2	2	100
Not mentioned	4	1	25

The cases of acute mediastinitis are of particular interest. In the five cases in which acute uncomplicated mediastinitis is given as the cause of death, autopsies were not performed. In all of the cases in which autopsy was performed the mediastinitis was found to be complicated by empyema, pericarditis or septic thrombosis of the azygos vein. This suggests that uncomplicated mediastinitis rarely causes death.

The mortality in the whole group and in the cases submitted to operation and those in which operation was not performed is shown in Table V. Of particular interest in this table are the results in the cases which were not operated upon. The mortality of 39.5 per cent in this group is the basis for the argument in favor of early prophylactic mediastinotomy in all cases of proved perforation of the esophagus.

TABLE V
SUMMARY OF SEVENTY-TWO CASES OF ESOPHAGEAL PERFORATION

	No. of Cases	Percentage
Operation performed	29	40.20
Operation not performed	43	59.70
Recovery without operation	26	60.04
Death without operation	17	39.50
Recovery with operation	18	62.06
Death with operation	11	37.90
Deaths	28	39.0
Recoveries	44	61.0

TREATMENT

In none of the articles reviewed has the importance of posture been emphasized. Since most of the foreign bodies are located in the upper esophagus and descent of infection into the mediastinum is the complication chiefly to be feared, the maintenance of the patient in the prone position with the foot of the bed elevated should be valuable both prophylactically and therapeutically. It should favor localization of the infection in the neck. Furthermore, in many cases the esophagus is so completely obstructed that copious fluid secretions overflow into the pharynx. Cough is usually so difficult and painful that aspiration of infected material into the lungs becomes a real danger. Posture should do much to prevent this complication. From the time that the patient is first seen until all danger of infection is passed, strict maintenance of the prone position should be enforced.

The actual surgical problem varies with the condition of the patient when he is first

seen. In general it is similar to that presented by acute appendicitis. Although it is well known that many cases will recover spontaneously, it is so nearly impossible to say which case will fall into which group that the greatest safety lies in immediate operation. If one waits to see, he will frequently have waited too long.

Marschik was the first to use a prophylactic mediastinotomy. He recommended its immediate use in every case of suspected perforation of the cervical esophagus. Von Hacker, von Eicken, Schlemmer, Killian, Kahler, Furstenberg, Neuhoff and almost all others who have written on the subject have concurred in this opinion.

Table VI

FORMS OF TRETMENT

I. Postural drainage
II. Prophylactic mediastinotomy
III. Drainage of cervical and mediastinal abscess:
  (a) Cervical mediastinotomy
  (b) Thoracic mediastinotomy
IV. Endoesophageal drainage
V. Drainage of pleural cavity
VI. Esophagotomy

Kahler emphasized the fact that it is important to note the location of the perforation at esophagoscopy so that operation may be done on the proper side. He reported three instances in which a prophylactic mediastinotomy was followed by descent of the infection in the opposite side. If one side of the neck is more tender, painful or swollen than the other, this should be the side chosen for operation. In doubtful cases a collar bilateral operation is advisable.

Wesselys advocated direct exposure of the perforation and packing of the area. Most writers have emphasized the fact that either emphysema or fever are indications for immediate operation. Kahler stated that a small amount of emphysema without fever can safely be watched, and also cited a case in which fever alone led him to perform a mediastinotomy. It later became evident that the fever had been caused by a beginning pneumonia.

In general it must be emphasized that in cases of suspected or early perforation of the cervical esophagus, the operation of prophylactic mediastinotomy is so safe and the dangers of delay are so great that immediate operation is always indicated.

Cervical mediastinotomy is effective to the level of the fourth dorsal vertebra, but for perforations below that the thoracic operation is required. This is so much more serious a procedure that it should be withheld until definite evidences of extending infection or localized abscess are present. Yet although it is a serious operation, it is frequently life saving. When infection has reached the mediastinum one must bear in mind the danger of death from pressure and the frequency of complicating empyema and pericarditis. By draining and decompressing the mediastinum and being ready to drain the pleura and sometimes the pericardium, one should be able to prevent death in some of these cases.

Localized cervical abscesses require local drainage, a procedure which Seiffert advocates performing through the esophagoscope. Kahler believed that this procedure should be reserved for abscesses deep in the thorax where direct approach is more difficult and dangerous. Abscesses in the upper mediastinum should be drained through the neck.

Tracheotomy is occasionally indicated for laryngeal or high tracheal obstruction, while cervical esophagotomy for removal of foreign bodies which cannot be extracted through the esophagoscope is still occasionally indicated, as is gastrostomy for those lodged close to the cardia.

SUMMARY AND CONCLUSIONS

Five cases of perforation of the esophagus by a foreign body and sixty-seven cases collected from the literature are reported.

An analysis of these seventy-two cases indicates that pointed objects are most apt to cause perforation. The foreign body became lodged in the cervical esophagus in 62.6 per cent of the cases and in the thoracic esophagus in 17.1 per cent. In 20 per cent the location was not mentioned.

In forty-three cases no operation was performed. In 60.04 per cent of these the patient recovered and in 39.5 per cent the patient died. In the whole series there were twenty-eight deaths and forty-four recoveries.

The literature is reviewed and the statistics of the authors cited are reported.

The consensus of opinion is that perforation of the cervical esophagus is an indication for immediate prophylactic mediastinotomy. In perforation of the thoracic esophagus, in which dorsal mediastinotomy would be required, expectant treatment until signs of serious infection are observed is indicated. The value of postural drainage in the prone position with the foot of the bed elevated is emphasized.

REFERENCES

Alpin, J. Collar mediastinotomy in complications due to foreign bodies in the esophagus. *Ztscbr. f. Hals,-Nasen-u. Obrenb.*, 28: 292, 1931.

Balacescu and Cohn. Cited by Starck, H.

Berry, A. Retro-esophageal abscess from self-traumatism. *Laryngoscope*, 34: 120, 1924.

Carmody, T. E. Discussion of paper by Orton, H. B.

Cerf, L. H. Safety-pin in the esophagus. *Arcb. Otolaryng.*, 24: 282, 1936.

Furstenberg, A. C. Acute mediastinal suppuration. *Tr. Am. Laryng., Rbin. & Otol. Soc.*, 1929, p. 210.

Guisez, J. Extraction of foreign bodies from the esophagus. *Bull. et mém. Soc. cbir. de Paris*, 22: 112, 1930.

Halmágyi, B. Foreign bodies in the esophagus and air passages. *Monatscbr. f. Obrenb.*, 63: 847, 1929.

Iglauer, S. Discussion of paper by Orton, H. B.

Jackson, C. Peroral Endoscopy and Laryngeal Surgery. St. Louis, 1915. Laryngoscope Company, Publishers. Chapter 18, p. 334.

Jackson, C. Foreign bodies in the air and food passages: charted experience in 1,115 cases at the Bronchoscopic Clinic. *Tr. Am. Laryng., Rbin. & Otol. Soc.*, 1923, p. 441.

Kahler, O. Treatment of perforation of the esophagus by foreign bodies. *Hals,-Nasen-u. Obrenarzt.*, 42: 257, 1936.

Kaloperopulos. Cited by Starck, H.

Killian, H. Operative treatment in complicated cases of foreign bodies in the esophagus. *Arcb. f. klin. Cbir.*, 122: 382, 1923.

Kindler, W. Unusual complications after injuries to the esophagus by foreign bodies and in other ways. *Ztscbr. f. Hals,-Nasen-u. Obrenb.*, 19: 3, 1927; abstr., *Arcb. Otolaryng.*, 6: 400, 1927.

King, E. Perforations of the esophagus, with report of six cases. *Tr. Am. Laryng., Rbin. & Otol. Soc.*, 1929, p. 329.

Kramer, R. Discussion of paper by Orton, H. B.

Lambert, A. V. S., and Berry, F. B. The mediastinum: paths of extension of infection from focus in mediastinum. *Arcb. Surg.*, 14: 261, 1927.

Lederer, F. L., and Fishman, L. Z. Prophylactic mediastinotomy for perforating esophageal foreign bodies. *Arcb. Otolaryng.*, 19: 426, 1934.

Lerche, William. Surgical treatment of suppuration in posterior mediastinum. *Surg., Gynec. & Obst.*, 32: 232, 1921.

Lynah, H. L. A series of foreign bodies in the bronchi and esophagus. *Tr. Am. Laryng., Rbin. & Otol. Soc.*, 1919, p. 285.

Manges, W. F. Roentgen diagnosis of foreign bodies in the esophagus. *Am. J. Roentgenol.*, 17: 44, 1927.

McGibbon, J. E. G., and Mather, J. H. Perforation of the oesophagus by swallowed foreign bodies. *Lancet*, 2: 593, 1935.

Marschik. *Verbandl. d. Vereins deutscb. laryngol.*, Kiel, 1914; *Wien. laryng. Gesellscb.*, March 4, 1914; *Gesellscb. d. Aertz.*, 1914, 1918, May 4, 1917; *Wien. klin. Wcbnscbr.*, 29: 805, 1916. Cited by Schlemmer, F.

Miller, J. W. Extraction of foreign bodies from the food and air passages by direct methods. *Arcb. Otolaryng.*, 16: 188, 1932.

Moore, I. Foreign bodies in the oesophagus and respiratory passages. *Lancet*, 2: 566, 1919.

Myerson, M. C. Perforations of the esophagus. *Ann. Otol., Rbinol. & Laryngol.*, 37: 545, 1928.

Orton, H. B. Mediastinitis following esophageal foreign body: report of cases. *Arcb. Otolaryng.*, 12: 635, 1930.

Schlemmer, F. Experience with esophageal foreign bodies in a series seen over a ten year period. *Arcb. f. klin. Cbir.*, 114: 37, 1920.

Seiffert, A. Relation between esophagoscopy and the appearance of emphysema following perforation of the esophagus. *Ztscbr. f. Hals,-Nasen-u. Obrenb.*, 19: 295, 1928.

Sheehan, J. E. Five recent bronchoscopic and esophageal cases. Med. Rec., 95: 361, 1919.

Starck, H. Significance of esophagoscopy. *Ztscbr. f. Laryng.*, 1: 81, 1909.

Ter-Oganesjan, M. Haemorrhages with foreign bodies in the esophagus. *Acta Otolaryng.*, 21: 116, 1934.

Vinson, P. P. Foreign bodies in the air and food passages: report based on 334 cases. *M. Clin. North America*, 16: 1471, 1933.

von Eicken, C. On an unusually large abscess of the posthypopharyngeal wall caused by foreign body. *Ztscbr. f. Hals,-Nasen-u. Obrenb.*, 14: 61, 1926.

von Hacker, V. Operative treatment of periesophageal and mediastinal abscesses, with special reference to collar and dorsal mediastinotomy. *Arcb. f. klin. Cbir.*, 64: 478, 1901.

Wesselys. Cited by Kahler, O.

Wright, A. J. Punctured wounds of the oesophagus. *J. Laryngol. & Otol.*, 49: 175, 1934.

Yankauer, S. Two cases of foreign body in the esophagus removed with the aid of the esophagoscope. *Tr. Am. Laryng., Rbin. &. Otol Soc.*, 1911, p. 305.

Zindel, L. Foreign bodies in the esophagus. *Deutscbe Ztscbr. f. Cbir.*, 148: 281, 1919. Cited by Schlemmer, F.

BENIGN TUMORS AND TUMOR-LIKE CONDITIONS IN THE TRACHEOBRONCHIAL TREE*

CHEVALIER JACKSON AND CHEVALIER L. JACKSON

Honorary Professor of Bronchology and Esophagology, Temple University School of Medicine

Professor of Bronchology and Esophagology, Temple University School of Medicine

PHILADELPHIA, PENNSYLVANIA

A BENIGN growth is one that does not metastasize and whose cells do not infiltrate among adjacent normal cells. Benign as applied to a growth in the tracheobronchial tree is not synonymous with innocent inasmuch as such a growth may cause death primarily by asphyxia, or secondarily by the suppurative disease caused by its obstruction to ventilation and drainage. Some of the usually benign growths are sometimes encountered in malignant forms.[1]

INCIDENCE

Prior to the advent of bronchoscopy benign growths of the tracheobronchial tree were regarded as exceedingly rare. This was due to the fact that at autopsy the small primary growth was not found; it was obliterated by the great mass of suppurative pathology secondary to its bronchial obstruction. Often in the last stages the growth itself was destroyed. These small tumors in the prebronchoscopic days were not encountered by the surgeon though he was often called upon to treat the secondary abscesses. The medical diagnosis in such cases was bronchiectasis and this was in some cases correct as to the secondary pathology. Bronchoscopy has shown that histologically non-malignant tumors are relatively common, though not quite so frequent as malignant growths.[1]

ETIOLOGY

Inflammation is an etiologic factor in some tumors, though just why a rounded tumor-like contour, instead of a diffuse fibrosis, should be a more frequent result in the bronchi than elsewhere has not been positively determined. We have attributed the frequency of the rounded contour to the cylindrical form of the bronchi together with their physiologic movements as a moulding factor. The mechanism somewhat resembles that of the pharmacist's pill roller.

The cause of granulomata in some cases is specific infection, for example tuberculosis, syphilis, spirochetosis, blastomycosis and actinomycosis. We have seen a number of cases of epithelialized pedunculated tuberculoma of the tracheobronchial tree in patients with long-healed pulmonary tuberculosis. We have seen a number of cases of hematoma of the bronchi and have attributed the capillary rupture to cough.

In a few instances we have found organizing hematomata, but this form of tumor is nothing like so common in the tracheobronchial tree as in the larynx. It may therefore be said that trauma is a lesser etiologic factor in the tree as compared to the larynx. This would apply to the trauma of movement, airblast, tobacco smoke and dusty atmosphere. We have seen, however, nonspecific granuloma of the bronchi in patients with occupational silicosis and anthracosis, and have regarded this due to silicotic trauma. In two instances we removed a granulomatous mass from the orifice of the right bronchus of men who had been gassed three and six years, respectively, prior to bronchoscopic discovery of the papillomata. In four other cases of papillomata of the bronchi there was nothing to indicate an irritant factor. There is some support for suspicion of an infectious factor in laryngeal papilloma[5] but none so far as we know, regarding

* From the Department of Broncho-Esophagology of the Temple University.

bronchial papilloma. Inflammatory obstruction may be a factor in the etiology of retention cysts; but air cysts are congenital, and of course teratomatous cysts are, though other factors may be concerned in the associated secondary changes.

PATHOLOGY

Our records include histories of the following tumors and tumor-like conditions: angioma, hematoma, adenoma, myoma, myxoma, papilloma, fibroma, fibrolipoma, lymphangioma, lymphadenoma, lipoma, ecchondroma, osteoma, chondrosteoma, retention cyst, amyloid tumor, aberrant thyroid tumor, specific granuloma and nonspecific granuloma.[3] Chondromata and osteochondromata may be benign but are prone to develop malignancy, and by sarcomatous or other changes, even metaplasia. Edematous polypi and other more or less tumor-like inflammatory growths are occasionally encountered,[3] and are of the utmost importance because of the atelectasis, drowned lung and suppurative conditions they produce.[1]

Histologically, most of these growths do not differ greatly from the same growths encountered elsewhere, but some of them are quite different. One of these peculiar growths is an adenomatous tumor that when first removed by the bronchoscopist was classed as adenocarcinoma by most of the histopathologists.[9] After these growths had been arrested in a number of instances by bronchoscopic removal and in other instances by Roentgen ray treatment, some histopathologists revised the classification and called the tumors benign adenomata;[2] other pathologists still regard them as adenocarcinomata of very low grade malignancy. To the clinician it would seem well for the present to classify them in the borderline group, that is, benign in some instances, malignant in others.[1]

In one of our cases we bronchoscopically removed a specimen from an endobronchial growth in a woman who was admitted almost moribund from profuse pulmonary hemorrhages. The hemorrhages were arrested by bronchoscopic insufflation of bismuth subnitrate. The histologist, Dr. Baxter L. Crawford, reported the growth adenocarcinoma. The tumor was arrested by irradiation and for twelve years there was no recurrence. Then rather suddenly there was an atelectasis of the right lower lobe. Bronchoscopy showed a tumor at the same location as before. A bronchoscopically removed specimen was regarded by Dr. Frank W. Konzelmann as a benign adenoma. Doctor Baxter L. Crawford, since the first report, had seen quite a number of similar cases and after much thought and study had come to the conclusion that the histologic appearances justified the name of benign glandular tumor. Comparing the sections of the last tumor with a slide of the first specimen of tumor bronchoscopically removed he found essentially the same histologic structure as in the first tumor. Further irradiation could not be given. Enough of the growth was removed to restore function to the atelectatic area. Two years later, fourteen years after the first tumor, called adenocarcinoma, was removed, the patient came in with an adenocarcinoma of the breast on the same (right) side.

One pathologically important peculiarity of benign growths in the tracheobronchial tree is that, though small, they set up diffuse destructive secondary changes in the lung out of all proportion to their size. In this there is an anatomic factor in the relatively small diameter of the bronchi, and a physiologic factor in the dependence of the resistance of the lung largely on ventilation and drainage.

The deeper parts of the tracheobronchial tree are practically sterile. The few organisms present are powerless against the defensive power of the lung so long as ventilation and drainage are normal.[1,6] When ventilation and drainage are stopped by a tumor occluding a bronchus the few bacteria present in the distal area become active; suppurative inflammation develops and becomes progressively worse. The suppurative stage often comes on quite

suddenly with an attack of influenza or other acute respiratory infection because of the inflammatory occlusion of the relatively small remaining by-pass for air at the site of the tumor.[6]

As discovered bronchoscopically, the obstruction of a bronchus by a tumor is of three types, mechanically similar to the valves in air pumps, namely, a stop-valve, a check-valve or a by-pass valve.[6,10] The stop-valve action is complete occlusion; it slowly results in atelectasis of the distal area as the air is absorbed by the circulating blood. The check-valve acts according to its set. With an ordinary air pump a reservoir may be pumped full if the check valve is set against outflow. If the check-valve is set against inflow the reservoir will be pumped empty by the same pump until a partial vacuum is created up to the limit of the power of the pump. In the lungs the set of the check-valve mechanism against outflow produces emphysema in the distal area. If the set is against inflow the lung is deflated by the pumping out of air, and atelectasis develops rapidly. We have seen with the fluoroscope a lobe become atelectatic in a few minutes when we displaced a tumor downward into the orifice of the corresponding bronchus, and have seen the lobe promptly expand again as the tumor was gently drawn upward. (Re-expansion would have required a longer time if the atelectasis had been of longer duration.) It should be added that atelectasis of this type has nothing to do with pneumothorax. Additionally, it should be stated as a pathologic fact that the obstructive type of atelectasis is also different in that suppuration soon develops in the atelectatic area if it is allowed to continue. It should also be emphasized that a peribronchial tumor by a compressive bronchial stenosis can cause a check-valve mechanism of the expansile type, producing an obstructive emphysema, as well as, later, a stop-valve mechanism, producing an atelectasis.

Air cysts communicating with the bronchi we have seen in a number of instances. Cystic growths of the bronchial mucosa were in some instances of the retention type and in others of the teratomatous type, containing hair and amorphous material. The larger cysts were associated with suppuration. Small cysts contained glairy mucoid material.

The wall of removed small retention cysts of the bronchial mucosa did not differ much histologically from those so common in the larynx.[5]

The histology of benign bronchial tumors has been extensively revised since the development of bronchoscopic biopsy. Further consideration of pathology and histopathology will be found by consulting the appended list of references. The bronchoscopic appearances of the living pathology, with abundant illustrations in black and white as well as in color will also be found in the works listed.

SYMPTOMATOLOGY

The symptoms depend upon the stage at which the patient is seen. In the beginning the patient is symptomless. A wheeze heard at the open mouth, not at the chest wall, is usually the earliest symptom and is almost always present later. The sound is quite similar to asthma, so much so in fact, that many of our patients had been regarded as having asthma.

Cough and slight mucoid expectoration appear as the growth progresses, usually becoming more troublesome when the tumor occludes the bronchus. Dyspnea independent of exertion does not occur from obstructive atelectasis of a lobe or even of an entire lung. In one of our cases a mass of epithelialized granulomata occupying the orifice of the right bronchus and causing obstructive atelectasis of the entire right lung did not produce dyspnea except on exertion. On the other hand, a histologically similar tumor mass at the bifurcation, which occupied part of both main bronchial orifices, caused intense nocturnal attacks of dyspnea, simulating asthma so closely that the patient had been treated for

months under this erroneous diagnosis. Complete relief of these symptoms followed bronchoscopic removal of the tumors.

Hemoptysis is a symptom of benign bronchial tumor and is quite constantly associated with adenoma. Strange as it may seem, benign growth as a diagnostic possibility is omitted in many of the textbooks in presenting the subject of hemoptysis.

DIAGNOSIS

Benign bronchial tumor as a clinical entity developed from incidental discoveries at diagnostic bronchoscopies. For this reason the first impulse is to dispose of the subject of diagnosis with advice to have a bronchoscopic examination. Though this is good advice it would not be good literature. Moreover, to follow it would not be good clinical procedure. Equally bad would be an attempt to make a diagnosis from the symptoms, because none is characteristic, let alone diagnostic.

Orderly procedure calls for: (1) anamnesis, with careful record of symptoms; (2) general medical examination with record of all physical signs (interpretation may well be postponed until later); (3) taking of specimen for serologic test and blood picture record; (4) repeated sputum examinations; (5) Roentgen ray examination; (6) diagnostic bronchoscopy; (7) bronchoscopic biopsy; (8) histologic examination.

In our cases the material and recorded findings thus obtained are taken to a *chest conference* at which the diagnostic problem is discussed by the internist, phthisiologist, roentgenologist, pathologist, bacteriologist, allergist, surgeon and broncho-esophagologist. A diagnosis, or at least a working diagnosis, is reached and plans are laid for further procedure.

Physical signs in case of uncomplicated, non-obstructive bronchial tumors are negative. Just as a growth reaches a size sufficient to cause the slightest degree of obstruction the physical signs are definite and of great importance. The following statements are based upon our clinical studies,[3,6] and were the sequel of our earlier studies of foreign body obstruction.[7]

The earliest of the physical signs of bronchial obstruction, whether caused by a tumor or anything else, is a wheeze heard at the open mouth. This had so often been mistaken for asthma prior to admission of the patient that in order to call attention emphatically to the necessity for differentiating it from a true asthmatic wheeze we called it the "asthmatoid wheeze."[11] Of course there is no need for the distinction where asthma is thoroughly studied, as in asthma clinics. We must not, however, lose sight of the fact that many of the patients found at diagnostic bronchoscopy to have tumors, benign as well as malignant, were allergic and had been treated for the allergic condition in some instances for many months. Many of these patients had nocturnal attacks of dyspnea due to the tumor and also to secretions accumulated during sleep, completing a misleadingly typical picture of asthma. In view of this experience, we feel justified in stating that the busy practitioner should be on guard against overlooking bronchial tumor in patients with a syndrome of wheezing, nocturnal attacks of dyspnea, and allergy. Furthermore, though we may be biased, we believe the records of the bronchoscopic clinic warrant the statement that every patient presenting a clinical picture of asthma requires a diagnostic bronchoscopy before it can be said that he has had the benefit of all that modern medical science can do for him.

Next in importance to the asthmatoid wheeze heard at the open mouth are the physical signs of by-pass valve obstruction, chiefly harshness of breath sounds distal to the tumor. Then come the physical signs of check-valve obstructions producing atelectasis and emphysema. These also are distal to the tumor. It is not necessary here to consider these signs, but it is necessary to say, and to say it emphatically, that when the physical signs show atelectasis or emphysema, bronchial tumor should be placed first among the diagnostic

possibilities. Diagnostic bronchoscopy is the only means for determining the character of the lesion or substance concerned in the check-valve mechanism.

Of diagnostic importance equal to that of the physical signs and the diagnostic bronchoscopy is the *Roentgen ray examination.* It is true that small endobronchial benign tumors do not cast a shadow, yet fluoroscopy is the best means we have for demonstrating the differentials in aeration present in all cases of check-valve obstruction, a form of obstruction which always raises the question of bronchial tumor. Moreover, in case of large benign growths causing stop-valve or check-valve compression stenosis the growth usually shows as a shadow in the roentgenogram and the compression of the bronchus can be demonstrated by insufflation of bismuth subcarbonate[1] or the instillation of lipiodol into the tracheobronchial tree.[8]

Bronchoscopic Examination. The technique of bronchoscopy and the bronchoscopic removal of tumors has been presented and elaborately illustrated elsewhere.[1,2,3,4]

Bronchoscopic biopsy is the only means known to medicine today for the determination of the character of a benign bronchial tumor. Histologic differentiation as among benign tumors is beyond the scope of this paper. It has been extensively discussed elsewhere.[1,2,3,4]

TREATMENT

The plan of treatment in each case in our clinic is discussed and often determined at the chest conference mentioned above in connection with diagnosis.

Large peribronchial compressive growths, if radical treatment is really required, are dealt with by external surgery. This applies particularly to epidermoid cysts causing suppurative destruction of pulmonary tissue. It is mechanically feasible bronchoscopically to penetrate compressed, uninvolved bronchial wall to reach a benign peribronchial growth, but the advisability of doing so is questionable. In our cases of air cyst, treatment was not indicated except when the interior was infected and suppurating. The large teratomatous cysts were associated with profuse suppuration and were operated upon by the surgeons who referred the patients for diagnostic bronchoscopy. One patient admitted with a diagnosis of bronchiectatic abscess was found after a number of bronchoscopic aspirations of enormous quantities of foul pus, to have a large epidermoid cyst; external operation was required for removal.

X-ray treatment externally applied is usually effective in glandular tumors, particularly lymphoma, adenoma, thymic and thyroid tumors. Bronchoscopically applied *physical therapy* has been useful in some instances.

Bronchoscopic Removal. Endobronchial benign growths of any size can be removed bronchoscopically. The *indications* for the procedure are any signs of bronchial obstruction or any suspicion that an unobstructive tumor will become obstructive. It is necessary to emphasize that when we say become obstructive we do not mean occlusive. The slightest sign of approaching obstructive atelectasis or approaching obstructive emphysema, no matter how small the area manifesting such a sign, calls for bronchoscopic removal, unless there are preponderant contraindications. To wait for occlusion would be a mistake. Apart from any of the indications mentioned in the foregoing, we believe any small tumor formation encountered bronchoscopically may well be extirpated because of its potentialities. Impaired drainage even of normal secretions is an important indication for removal of any tumor however small. Apart from obstruction by bulk, a tumor usually has no cilia; drainage efficiency is proportionately impaired.

The most important *contraindications* are a strongly positive serologic test, a high, irreducible hypertension, or any organic disease that of itself lessens the patient's life expectancy. Less serious degrees of hypertension are not a contraindication. Hemorrhage unless exsan-

guinating is not a contraindication. In some cases it is advisable to postpone removal of a growth until after transfusion and other appropriate methods have tided the patient over the anemic danger point. On the other hand, bronchoscopy may be indicated for hemostasis in some such cases.

The *technique* of introduction of the bronchoscope is so well established as to require no presentation here. In most instances forceps are the preferable instruments for bronchoscopic removal of benign tumors. The two forms most useful are the cupped forceps of tubular mechanism and the sharp-cornered basket punch forceps. The latter have a clean cutting, shearing mechanism that must be used with utmost precaution and precision to avoid danger of invading normal tissues; they are needed only in case of chondroma, osteoma and other very firm growths. The cupped forceps are quite safe if used with delicacy and keenness of touch. Plain slender-jawed forceps are useful chiefly as dilators.

In case of small pedunculated tumors the growth may well be completely removed, using great care to avoid any damage to basic normal tissues. In case of large growths or sessile small growths, it is usually preferable to know the histologic structure of the tumor. Hence, in these cases bronchoscopic biopsy is a proper preliminary.

Small inward projecting retention cysts should be amputated bronchoscopically. Small mucous cystic cavities should be evacuated by inserting slender forceps jaws closed and then expanding them to distend the orifice. If reaccumulation occurs repeatedly one edge of the orifice may be nipped out. Non-specific granulomata should be nipped off superficially. Specific granulomata, including fungoid growths, may be removed; but removal of epithelialized syphiloma and tuberculoma may be contraindicated by general conditions. Papillomata are usually multiple; they should all be scalped off superficially, including every little bud, but neither forceps nor caustics should be used on the base. Radical extirpation is useless and unnecessary and involves needless risk. New growths may appear in new locations no matter how radical the primary extirpation may have been. Superficial scalping with the cupped forceps will remove all papillomatous tissue yet leave all normal tissues unharmed.

The word scalping is used here in contradistinction to cutting, biting or even nipping. The cupped jaws and delicate touch enable the operator's fingers to distinguish normal tissue by its resistance, after the jaws have passed through papillomatous tissue. When this resistance is felt the forceps are not compressed further, but are gently withdrawn, allowing the two cups to slide off the normal base without injuring it.

Pitfalls in diagnosis are due to delay or failure in availing one's self of the information obtainable by diagnostic bronchoscopy. Some of the pitfalls in external operation can be avoided by coöperation with the bronchoscopist. Pitfalls in bronchoscopic removals of benign growths are almost all avoidable by conservatism, caution and utmost delicacy of touch.

PROGNOSIS

Some benign endobronchial tumors are fatal, primarily by asphyxia or secondarily by the sequential suppurative disease of the lung. Almost all benign endobronchial tumors and tumor-like formations can be removed bronchoscopically; and the prognosis as to life in such cases is good. Recurrence is a possibility, especially in cases of lymphoma and papilloma; in papilloma and some forms of granuloma new growths may appear at new locations after removal of the primary growth. In such cases reappearances usually cease after repeated superficial removals. The prognosis of benign adenoma is good provided the patient be seen occasionally in order to detect and treat recrudescence or recurrence early.

REFERENCES

1. Jackson, Chevalier, and Jackson, Chevalier L. Bronchoscopy, Esophagoscopy and Gastroscopy. Philadelphia, 1934. W. B. Saunders Co.
2. Jackson, Chevalier L., and Konzelmann, Frank W. Bronchoscopic aspects of bronchial tumors with special reference to so-called bronchial adenoma. *J. Thorac. Surg.*, 6: 312 (Feb.) 1937.
3. Jackson, Chevalier, and Jackson, Chevalier L. Benign tumors of the trachea and bronchi with especial reference to tumor-like formations of inflammatory origin. *J. A. M. A.*, 99: 1747–1753 (Nov. 19) 1932.
4. Jackson, Chevalier L., and Konzelmann, Frank W. Bronchial carcinoma. *J. Thorac. Surg.*, 4: 165 (Dec.) 1934.
5. Jackson, Chevalier, and Jackson, Chevalier L. The Larynx and Its Diseases. Philadelphia, 1937. W. B. Saunders Co.
6. Jackson, Chevalier. The mechanism of physical signs in neoplastic and other diseases of the lung with especial reference to atelectasis and emphysema. *J. A. M. A.*, 95: 639–644 (Aug. 30) 1930.
7. Jackson, Chevalier. Mechanism of physical signs, with especial reference to foreign bodies in the bronchi. *Am. J. M. Sc.*, 165: 313–320 (March) 1923.
8. Jackson, Chevalier L., and Bonnier, Maurice. Technic of bronchography. *Ann. Otol., Rhinol. & Laryng.*, 46: 771 (Sept.) 1937. Also in *Proc. Am. Bronch. Soc.*, 1937.
9. Jackson, Chevalier L. Adenoma bronchiale. *Il Valsalva*, 1937.
10. Jackson, Chevalier. Obstruction bronchique *Bull. et mém. Soc. méd. d. hôp. de Paris*, 54: 1583–1584, 1930.
11. Jackson, Chevalier. A new diagnostic sign of foreign body in trachea or bronchi, the "asthmatoid wheeze." *Am. J. M. Sc.*, 156: 625 (Nov.) 1918.

The man who consults us, apparently at his wits' end from the nagging of a jealous, hen-pecking wife, may very possibly also have an ulcer gnawing at his duodenum.

From—"Disease and the Man" by Roger F. Lapham (Oxford).

BOOK REVIEWS

La Thérapeutique du Nourrisson en Clientèle. By Pierre Nobécourt and Marcel Maillet. Third edition. Paris, 1937. Maloine.

The third edition of this book covers briefly the entire field of pediatrics in the regular orthodox manner. There are no illustrations. The authors give the French viewpoint of treating diseases and handling situations as they arise in the growing child. No material differences are apparent between the American way and the French methods, so far as this particular volume goes, although some of the drugs used and the manner in which they are used may be new to some of us.

Précis D'Urologie. By Félix Legueu and Edmond Papin. Second edition. Paris, 1937. Maloine.

This is the second edition of the précis. It is a book of slightly over 700 pages containing 511 illustrations most of which are schematic. The authors are internationally known urologists who have written this outline of urology mainly for their students. It is therefore very simple and concise, as a primarily didactic treatise should be.

Most of the subject matter is not debatable; it is just the common sense of urologic practice. However, even for such a concise outline certain omissions should be criticized. The new advances in chemotherapy as applied to urologic diseases should be of great interest to students who go out into general practice, but they are not even hinted at. The field of cystometry as a diagnostic aid is not mentioned and the notable advances in the technique of transurethral operations are not adequately discussed.

An outstanding feature of the book is the detailed description of renal function tests, particularly the tests using urea elimination as an index.

There is no bibliography, but an alphabetical index as well as a tabulation of contents.

Les Diagnostics Chirurgicaux au Lit du Malade. By M. Barthélemy. Two volumes. Paris, 1933–1938. Maloine.

These two volumes on Bedside Surgical Diagnosis by M. Barthélemy, Professor of Surgical Pathology at the Medical Faculty of Nancy, France, consist of didactic lectures and bedside discussions over a period of years in surgical teaching to undergraduate students. The author covers a great number of surgical entities from the diagnostic point of view and does it well, clearly and concisely. Both books are illustrated by the author. The drawings are helpful and well done.

Volume I, just published, is much larger than the second volume issued in 1933 and the subject matter is more diversified. This work may be used as a reference for students and practitioners interested in the diagnosis of their surgical problems.

Surface and Radiological Anatomy for Students and General Practitioners. By Arthur B. Appleton, M.A., M.D., William J. Hamilton, M.D., F.R.S.E., and Ivan C. C. Tchaperoff, M.D., D.M.R.E. Baltimore, 1938. William Wood & Co. Price $5.50.

What would we not have given to have owned this book when as a student we studied anatomy nearly thirty-five years ago? The authors truly have made the dry topic of anatomy interesting and engrossing. They have ventured beyond the usual accepted texts on surface anatomy, and to the time honored methods of physical examination they have added the employment of radiology. A knowledge of the normal appearance is indispensable as a background to the proper utilization of *x*-rays for clinical purposes. By an extensive use of excellent illustrations they have supplied a means of coördinating radiologic anatomy with those features which can be determined by external examination and with those details which can be determined only by dissection.

We could go on at length telling the special features of this work. Instead the reader will have to accept the statement that it is the best work of its kind we have met with to date.

We know a young man who begins the study of medicine this year. We intend giving him our copy as a present. We know he will be the

envy of his fellow students, and, also, that many of his classmates will, in turn, purchase copies. For it should have a wide appeal to medical students, beginners in radiology, and many surgeons.

MATERNAL CARE. COMPLICATIONS. Edited by F. L. Adair, M.D. Chicago, 1938. The University of Chicago Press. Price $1.00.

This small book of only 95 pages is worth its weight in gold. It deals with the principles of management of some serious complications arising during the antepartum, intrapartum, and postpartum periods.

A year ago The American Committee on Maternal Welfare, Inc., published a small book entitled "Maternal Care," which was extremely well received. This second volume attempts to present the essential facts relative to the three major causes of maternal mortality: toxemias of pregnancy, obstetrical hemorrhages, and puerperal infection. These were prepared by Drs. R. D. Mussey, P. F. Williams, and F. H. Falls, respectively. We learn that copies of the three manuscripts were then sent to the various directors of the Committee, who reviewed the papers and resubmitted them to the editor with their suggestions and comments. The ideas expressed are therefore truly a cross section of the best obstetrical minds in the country.

Will physicians, by and large, trouble to pay a dollar and order copies? It's too bad some rich foundation or public spirited citizen overburdened with wealth could not distribute about 75,000 copies where they would do the most good. For if the majority of American doctors, especially those who more or less do the obstetrics in this country, were to read these booklets, we are sure better obstetrics would result, and maternal mortality would show a downward curve.

Dr. Adair and his co-workers are to be congratulated on doing a splendid practical work.

A TEXTBOOK OF GYNECOLOGY. By Arthur Hale Curtis, M.D. Third Edition. Philadelphia, 1938. W. B. Saunders Company. Price $7.00.

In reviewing the first two editions of Dr. Curtis' work on Gynecology, we had nothing but praise for his efforts and their result. This third edition merits the same report. We feel it was and is one of the best textbooks of modern times.

The new volume has been entirely reset and contains 318 illustrations, chiefly by Tom Jones. Eight preliminary chapters have been added, the subject matter has been entirely rewritten, and much new material is included. Anatomy (omitted from the first edition), physiology and the endocrine glands are given detailed consideration.

We still feel that this compact work of 603 pages, with its adequate bibliography and complete index, is one of the best textbooks on gynecology by an American author.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY. 18th Edition. By W. A. Newman Dorland, A.M., M.D., F.A.C.S. Philadelphia, 1938. W. B. Saunders Company. Price $7.50.

The reviewer has known this volume for years as "Dorland's Medical Dictionary." And now welcomes it as it comes to the profession in its eighteenth edition, compiled by Dr. Dorland with the collaboration of Dr. E. C. L. Miller.

How does one set about reviewing a dictionary? What does he say? Should he look to see if some new pet word has been included? And if he finds it missing, should he damn the entire work?

We hardly think that a good method. This is a marvelous book—one that fulfils many requirements and answers every need that a medical dictionary could be asked to supply. The modern physician should have a medical dictionary at his right hand. If he lacks one, or could use a new one, this volume by Dorland can be recommended without reservation.

It has been brought out in two versions, with stiff or flexible binding, is 1607 pages in length, and contains 942 illustrations (some in color) and 283 portraits.

L'ANGINE DE POITRINE. By Vital Lassance. Paris, 1938. Maloine.

It is unfortunate that the tendency to consider angina pectoris a disease entity continues to prevail. The author of this work understands this, but the fact that he calls his book "L'angine de poitrine" adds to the confusion. The work contains a good description of the

anginal syndrome, its causes, recognition and treatment, so well as of coronary thrombosis. The French and American ideas are very similar.

There is much repetition in the book, partly because the anginal syndrome is first described by itself and then in relation to coronary insufficiency. Citation of other authorities forms the bulk of the sections on pathology.

The reviewer also thinks that the author has emphasized too much the angina-like syndromes, particularly aortalgia. He makes positive statements about inflammation of the aortic plexus in syphilitic aortitis about which there is no proof. More and more evidence is accumulating that aortic pain is really coronary pain caused by syphilis of the coronary ostia.

With these exceptions "L'angine de poitrine" is an excellent presentation of the subject—Heart Pain.

MANAGEMENT OF FRACTURES AND DISLOCATIONS. By the Staff of the Fracture Service, Massachusetts General Hospital, Boston. Philip D. Wilson, M.D., Editor. Philadelphia, 1938. J. B. Lippincott Company.

The reader who is a surgeon or a general practitioner who handles fractures and dislocations should own a copy of this book. We believe he will read it carefully, and will do little or no skipping. For the staff men of the fracture service of the Massachusetts General Hospital have offered us a unique and most satisfying work. It differs from the average run of books on the subject in that the authors have based their material upon the actual study of cases and the analysis of the results of treatment. We are informed that the "conclusions [of the book] are fortified not only by the observation of the patients during their stay in the hospital, but by the actual examination of the same patients a year or more after discharge from the hospital with critical appraisal of the results in terms of anatomic restoration, functional recovery and working ability or earning power."

The study has to do with a group of 3,985 patients with 4,399 fractures and dislocations, treated over a period of seven years by the fracture staff at Massachusetts General Hospital in Boston.

The book is 1,036 pages long, has 1,419 illustrations, of which 1,192 are line tracings of roentgenograms in case reports, and an ample index.

We cannot go into detail. Suffice it to say that all fractures and dislocations are given adequate consideration. The book is beautifully done. The inside cover and first page give the keynote, with two skeletons in color, depicting the various fractures, and the cases reported.

We would like to see books on other topics follow the plan and scope of this one. The men of Boston who made this marvelous work possible are to be congratulated. This is certainly a book that can stand forth proudly on any library shelf.

A SHORT PRACTICE OF SURGERY. By Hamilton Bailey, F.R.C.S., and R. J. McNeill Love, M.S. Fourth Edition. London, 1938. H. K. Lewis & Co. Price 24 shillings.

This work has appeared in three previous editions, and now a fourth edition is being offered to the profession. As in the previous editions, the authors have included only such material as is completely up to date, and yet generally acceptable. Advanced surgery and rare conditions are "relegated to small type." The authors believe "one picture is worth a million words," and the illustrations, of which there are 818, 109 in color, truly lighten and applify the text. Some former illustrations have been replaced with new ones, and fifty-five additional figures have been added.

A new feature of the fourth edition is the inclusion of short historical notes.

An excellent book, especially for the beginner in surgery, the surgical intern, and the resident fellow.

SURGICAL PATHOLOGY. By William Boyd, M.D. Fourth Edition. Philadelphia, 1938. W. B. Saunders Company. Price $10.00.

The first edition of this recognized work appeared in 1925. The second edition followed in 1929, while another edition appeared in 1933. In this, the fourth edition, new sections have been added, others have been rewritten or changed, and an introductory chapter present in the first two editions, but omitted from the third, has been reinserted. Over twenty additional subjects have been included. to this edition. The new material deals with the experi-

mental production of cancer, the etiology of tumors, the etiology of appendicitis, anaerobic streptococci in puerperal sepsis, and other subjects too numerous to mention here. Dr. P. H. T. Thorlakson of Winnipeg rewrote the chapter on Diseases of the Rectum.

This book (886 pages) has 476 illustrations and fifteen colored plates, references at the conclusion of each chapter, and an index.

Any scientific work that goes through four editions in thirteen years' time needs no other recommendation.

THE PRINCIPLES AND PRACTICE OF MEDICINE. Originally written by the late Sir William Osler. Revised by Henry A. Christian, M.D., LL.D., S.D., F.R.C.P. Thirteenth Edition. New York, 1938. D. Appleton-Century Co. Price $9.00.

Osler wrote the preface to the first edition of this time-honored work at Johns Hopkins Hospital, Baltimore, on January 1, 1892. The book caught on immediately and since then has enjoyed a large distribution all over the world. It went through many revisions, and after Osler's death, the ninth, tenth, eleventh and twelfth editions were revised by Thomas McCrae of Philadelphia. McCrae died, and now we are offered a thirteenth edition by the Hersey Professor of the Theory and Practice of Physic at Harvard, Dr. Henry A. Christian. The publishers could not have selected one better equipped to carry on this work.

A book of this caliber, observed in most physicians' libraries, needs no detailed review. For over forty-five years it has stood in the front rank of books on this subject. It still leads. We can do no better than quote from parts of Dr. Christian's preface: "A pupil of Osler, a friend and colleague of McCrae, I welcome the opportunity of continuing the medical traditions of this book, which for so many years had the guidance of these master clinicians. In this, the Thirteenth Edition, my purpose has been to continue its traditions, attuning them to such newer knowledge in the field as recently has developed. To this end there has been rearrangement, rewriting and the addition of new material."

The book is 1,424 pages long, amply indexed, but devoid of illustrations. Dr. Christian, who in preparing the revision, tells us he "had the assistance or criticism of no one," has done a masterly piece of work. No doubt, it still will continue to be the student's medical Bible.

LA BLENNORRAGIE CHEZ L'HOMME ET CHEZ LA FEMME. Méthodes Actuelles de Diagnostic et de Traitement. By Dr. Pierre Barbellion. Second Edition. Paris, 1937. Maloine.

The second edition of this work by Dr. Barbellion is a sound and modern presentation of the current French viewpoint on gonorrhea. Although there is nothing particularly new in the presentation it is based on long personal experience of a splendid clinician who is more familiar than the average with the research problems of this unconquered disease. There are shrewd discussions as to diagnosis of gonorrhea and pseudo-gonorrhea by laboratory methods. The author doubts the value of vaccines and chemotherapy. He gives practical directions for therapeutic procedures, and sincere judicious opinions on the value of heat serums and so forth. To one who reads French easily this is a valuable book. The importance of this subject, the high character of the author's presentation are attested in a preface by Professor F. Legueu.

OUTLINE OF ROENTGEN DIAGNOSIS. By Leo G. Rigler, B.S., M.B., M.D. Atlas Edition. Philadelphia, 1938. J. B. Lippincott Co. Price $6.50.

This excellent book is made up in two formats, one, in which the 254 illustrations and x-rays are grouped in atlas fashion with numerous references and cross references in the text (this is the one reviewed), and the other a student edition, from which the atlas section has been omitted (priced at $3.00). We urge the purchase of the Atlas Edition—it is worth it.

It was not long ago one graduated from a medical school without any knowledge of roentgenology. Some ten years ago Dr. Rigler prepared a series of lecture notes in mimeograph form for his students' use. These notes, revised and expanded, have been used as a supplement to didactic lectures to undergraduates, as a text in teaching extension courses to general practitioners, as a study outline for graduate students, and, by radiologists, as a synopsis for lectures. This volume is the result of numerous requests for a more permanent format.

The work is divided into eleven sections: General Principles of Roentgen Diagnosis; Bones and Joints; Diseases of Spine and Spinal Cord; Skull and Its Contents; Thorax; Digestive Tract; Gall-bladder; Abdomen; Urinary Tract; Female Generative Organs; and Miscellaneous. One is especially directed to the paragraphs in which an effort has been made to present, as definitely as possible, the relative value of Roentgen diagnosis in each disease process.

The drawings by Jean E. Hirsch which illustrate the diseases of the bones and joints are especially worth notice. The reproduction of the roentgenograms is beautifully clear and sharply defined.

The author's style is clear and to the point. The bibliography leaves nothing to be desired. There is an index. Medical students and physicians who feel they need an up-to-date, authoritative work on this subject need look no further.